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The Jackson Lecture.[1]

THE HISTORICAL BACKGROUND OF MEDICINE.

By HUGH MCLELLAND,
Brisbane.

IN the search for truth a study of relevant history is always useful and as a general rule necessary. The more we concern ourselves with fundamental truth, the more we find it necessary to study the past. This becomes clear when we realize that the truth is actually that which in the longest view succeeds. The long view is the historical one. It is not given to us to see far into the future; but the general trends of success and failure are often visible lines that may be carried forward, giving us a clue to success in the future. At the very beginning, however, we are confronted with a serious difficulty. In following the evolution of doctrine, creed and method in every phase of human activity, we find still with us a trace and sometimes much more than a trace of almost every superstition and heresy that formerly confused and divided mankind. All the old errors of method and thought that were supposed to have ended in the blackest disgrace are still here. Are we to say that we have just not had time to eliminate them; that they are on their way out and only linger on as archaic traces like our branchial arches; or that they survive because they, like antitheses, still contain an element of the truth? At least we are able, by studying history, to pin down some of the worst mistakes by showing the faults of method and of reasoning which produced them, and by demonstrating that the apparently good results of error have been illusory and have not stood the test of time. On the other side of the ledger is the true opinion which is backed only by faulty reasoning. Many of the beliefs and judgements of children are true enough, though it is unusual for a child to give reasons that appear valid to an adult. There are indeed many wise people who refuse to give reasons when they are sure that they are right. On the other hand, there is the case of the High Court—the ultimate in legal wisdom. Yet its members, though they occasionally give a unanimous judgement, seldom give identical or even compatible reasons.

As we follow the thread of truth in history—a crooked thread and broken in many places by the foolishness of the wise, by the logical errors of logicians and even by the clumsiness of good technicians—we find it hard at first to resist an impulse to feel immensely superior in the possession of the great body of knowledge which is our heritage today—as if it were not a heritage at all, but our own individual achievement! The smug feeling soon gives way to gratitude for those oldtimers who by their ceaseless efforts and sufferings laid the foundation on which we build today. Indeed, the greatest admiration of the ancients comes from those who have studied them most and know most about them. Nothing that we find in history is irrelevant; not even the patient exploration of such dead ends as the scholastic logic of the Middle Ages, or even such wills-o'-the-wisp as the elixir of life and the philosopher's stone.

In the beginning the practice of medicine and the practice of magic were closely related. In primitive societies the physician or medicine man was a magician and relied on the black art for his results, although the common sense of tested experience was not entirely lacking. Sir James Fraser in his famous book[(1)] has analysed the logic of magic with numerous examples. He finds that all magic is "sympathetic magic", and the belief is that all events or objects or agents that produce in the observer the same feeling are related or even identical. This sympathetic magic is then either "homœopathic" magic or "contagious" magic. In the former case the belief is that like produces like. You can cause rain to fall by producing the appearance of rain, for example by pouring

[1] Delivered at a meeting of the Queensland Branch of the British Medical Association on September 3, 1948.

water through a sieve from the tree top. You can bring on a thunderstorm by beating a drum. You can cure your sick friend by simulating his pain and after applying some imagined remedy immediately resuming your normal health. The foster mother can establish a true blood relation with her adopted son by mimicry of the act of giving birth to him. In contagious magic the relation of likeness is replaced by one of contiguity. Objects, events and agents which are contiguous in space or time, being fused in the one experience of the observer, are held to be related or identical. The power for good or evil that inheres in any person or object can be transferred by contact. A little reflection on the logic and method of contagious magic will show that it is bound to give some results. In fact its logic is precisely that of the reflex and the conditioned reflex which are the product of causality in Nature coupled with the contingency of history. In the case of the reflex it is the history of the species and in the case of the conditioned reflex it is the history of the individual. The primitive medicine man then recognized both the causal sequences of Nature and the contingencies of history. It is not until our own day in the work of Pavlov, Sherrington and the psychologists of the *Gestalt* school that the integration of such disparate causal factors as law and accident comes to be studied, and it is recognized that contingency may be roped in within the ring of natural law and held there so long as the conjunction is useful. But the medicine man had no conception of natural law as we understand it today. For him nothing happens of necessity, but by the intervention of demonic agents who can be compelled by the right word or ritual to "do their stuff" at the appropriate time and place. Even the sun rises daily by the repeated operation of the will of some supernatural agent; or else the sun is itself alive. The invariable sequences of Nature are then in this primitive view nothing but the good and bad habits of the gods and demons.

The social standing of the medicine man is of interest to us. He was, of course, greatly feared and on the whole hated rather than loved by the tribe, just as the modern medicine man may be hated and feared as a member of a powerful sect, though he may be loved and trusted by a few. The obvious way to deal with such a dangerous power was to rope it in and enlist it in the service of the tribe. What could be better than to employ and to pay the magician and thus hold him in the power of the tribe to do its bidding? These conditions were sufficient to induce the tribe to have its official medicine man, and no doubt similar feelings in society today account for the priority which the medical profession enjoys in schemes of nationalization. The modern medicine man is still held to have the secret of the magic word and gesture which have power of life and death. The power of word and gesture, so real to the ancients, is still with us almost as great as ever, though fewer people believe in magic. In the beginning speech was no more than a system of sounds and cries whose use was to influence the environment, especially the environment consisting of people and animals, rather than to convey information. In law courts and parliaments, I believe that that is still its chief function. These sounds, exclamations and cries, with their accompanying gestures, were the first words. They denoted objects or else they were commands or threats. With them it was possible to influence behaviour and to get what one wanted. Words and gestures were very successful. They had power. They got results. The environment was duly impressed. It was inevitable that power should be attributed to the word itself—the mere sound of it. It was inevitable too that the word and the object denoted by the word should come to be identified in the mind, and that the mere sound of the word should be given all the attributes of the object which it denoted. Moreover, since all living things remember, words and objects are recalled together in the mind. The mind is such a busy integrator that the word and object are remembered together and fused in one experience with their whole context—that is to say, the object and its environment as well. It is unfortunate for truth and truthfulness that the object and its environment differ so much. They are, in fact, generally set off against one another in the best dialectical fashion. Thus the word has almost from the beginning a double meaning—two meanings probably incompatible with one another. Common old words have had so many contexts, taken part in so many events, that they are actually large slices of history. They can therefore, as Humpty Dumpty knew, be got to mean almost anything if you just treat them well. Such is their experience. The transition of the word as sign to the word as symbol is then quite easy. The characteristic of the word used symbolically is that it no longer merely points and commands. It strikes attitudes. It behaves like a living thing—well or ill. It has habits and a capricious will of its own. The baby quite early gets the idea of the omnipotence of the word. Its first words are commands that are obeyed in great haste, you will agree. The great Origen, one of the master minds of the early Christian era, was impressed by the magic of words. That there were really magic spells he had no doubt. And it was for him not a matter of indifference whether you called to God by His Latin, Greek or Hebrew name, for the words were not the same. Origen believed, too, that magic spells lost their power when translated.

With the introduction of symbolism into language, the verbalism which characterized the earlier medical, philosophical and theological writings was, I suppose, inevitable. The Greeks were great verbalists and went very far both in truth and in error by merely exploring and exploiting words, for words are often great chapters of history crammed with events. And the Greeks as often as not assumed that a word, being true in a certain context, was equally true in every other context and shade of meaning that it had picked up in its adventures. A famous and rather amusing example is provided by Asclepiades when he remarked that "Nature would as soon kill as cure a man". He was referring to the *vis medicatrix naturæ* with which Hippocrates had glorified Nature. Centuries later Galen abused Asclepiades for his irreverence towards the great master. It is clear, however, that there are here two natures. The nature of man will restore him to health. The other nature is the universal Nature, which includes besides the fruits of the earth and warm sunshine, the ice-age, droughts and famines, earthquakes, rats, vermin and microbes. This Nature will kill a man as soon as cure him. The word "Nature" is here a symbol and not a sign. It points to nothing in particular, but to many diverse things, and beyond them to a fictitious entity with a will and power. It is equated sometimes with God and sometimes with the Devil. Nevertheless, although the Greeks fooled themselves and one another with their equivocations, they were able to exploit mines of wisdom locked in the words themselves. After all, this was an historical approach, for the meanings of words lie in the history of words, which is the history of other times.

From magic and priestly medicine and the cult of Aesculapius to the age of Hippocrates is a tremendous leap. Hippocrates, philosopher and physician, combined in his person all the virtues that could be ascribed to the greatest physician. He was born at Cos about 459 B.C. and is seen against a background of Ionic philosophy. He took from the philosophers the notion of the four elements: earth, air, fire and water. These immediately gave him the four qualities: dry, cool, warm and moist. Hippocrates then found that there were four humours, blood, phlegm, yellow bile and black bile, and four corresponding temperaments. The number four was evidently very useful to him, for his concepts numbered four fours, or four sets of dialectical pairs. It will be noticed that all these entities are observables and can be apprehended by the senses. Hippocrates started thereby with a set of mental instruments with which he hoped to exclude metaphysics. Actually he could not succeed in this and was forced to invoke the *vis medicatrix naturæ*, which of course remained a mystery. Nevertheless his method remained one of acute observation and study and accurate description and the painstaking collection of all observable data. It follows that there is very little mere verbalism, and that slick deductions from facile generalizations are ruled out and all inferences can be tested by further observations. A great deal of the Hippocratic body of knowledge is,

therefore, valid still, and his descriptions are as good today as ever they were and serve as models of accuracy. There were faults, of course. There was a sad lack of anatomical and physiological knowledge, and there were no controlled experiments. However, the method of careful collection of data laid the foundation on which inductions could be built. Actually, there were no notable inductions in this phase. We shall presently consider what were the limiting factors in Greek science and philosophy, for we have to account for the fact that there was no real progress after Hippocrates for many centuries. The Hippocratic spirit as distinct from the method is expressed in the famous Hippocratic oath, which in some form is still the categorical imperative of every good physician. It expresses a religious feeling of duty and obligation and a devotion to others without thought of self. There is a complete absence of demons, amulets, charms and miracles. There is an implicit recognition of natural sequences and of contingency in the affairs of men. Hippocrates wrote industriously and travelled widely. Happily a great many of his writings are readily available to us. In his "Airs, Waters and Places" we get a close glimpse of the man and his thought. He went everywhere and wrote what he saw. Thus, characteristically, he deals only with observables. Although he takes plenty of time and uses plenty of words to explain, he still keeps to what he sees or senses. His account of the Scythians is typical. "The Scythians", he says, "are the reverse of prolific, and Scythia breeds the smallest and the fewest wild animals for winter is perennial and the wild animals are such as find shelter under ground." Of the men he says that "neither bodily nor mental endurance is possible where the changes are not violent. For these causes their physiques are gross, fleshy, showing no joints, moist and flabby and the lower bowels are as moist as bowels can be. For the belly cannot possibly dry up in a land like this". The dry and the moist are for Hippocrates primary qualities and therefore of very great interest. So he pursues the subject of moisture. "I will give clear testimony to their moistness", he continues. "The majority of Scythians, all that are Nomads, you will find have their shoulders cauterized . . . simply because of the moistness of their constitution. For owing to their moistness and flabbiness they have not the strength to draw a bow or to throw a javelin. . . . The girls are wonderfully flabby and torpid in physique. A constitution of this kind prevents fertility. The men have no desire for intercourse because of the moistness of their constitution and the softness and chill of their abdomen, which are the greatest checks on venery. In the women the causes of barrenness are fatness and moistness of their flesh, which are such that the womb cannot absorb the seed. For neither is the monthly purging as it should be, but scanty and late, while the mouth of the womb is closed by fat and does not admit the seed. These are the causes which make the Scythian race infertile. A clear proof is afforded by their slave girls. These, because of their activity and leanness of body, no sooner go to a man than they are with child." Presumably, the slave girls are warm and dry, not cold and moist. However, as though not quite satisfied that he had explained everything, he gave a further reason for male sterility. The men suffer from swellings of the joints and sores on the hips because they are always astride their horses. "They cure themselves in the following way. At the beginning of the disease they cut the vein behind each ear. When the ensuing faintness has passed off some are cured and some are not. Now in my opinion by this treatment the seed is destroyed. For by the side of the ear are veins to cut which causes impotence and I believe these are the veins which are cut."

From Hippocrates to Galen is a jump of more than five centuries. In that period there was not, however, much progress in medicine. Aristotle (384 B.C.), the Alexandrians Herophilus and Erasistratus, and the Roman encyclopædists Varro, Pliny and Celsus, were perhaps the greatest names. Aristotle's day, however, was not to dawn until the Middle Ages, although he was well known and widely read in his own time. The works of Herophilus and Erasistratus probably went up in smoke when Julius Cæsar's soldiers burned the great library in Alexandria. This was a great loss, for in Alexandria there was the beginning of a sound physiology and anatomy founded on experiment and dissection. What we know of their work is inference only, from references and quotations in contemporary and later writers. The great Celsus had not very much influence in his own time and had to be rediscovered after the Middle Ages. However, in these centuries after Hippocrates, Greek learning and culture were widely disseminated. After the wars of liberation, in which Ionia was freed of the Persian yoke with the help of Athens, the Athenians occupied the Ionian islands and founded the great Athenian empire. It was not a

TABLE I.

Ionia.

The Iliad	Though not Ionian the Iliad is said by Gilbert Murray to have been the grammar of the Ionian youth as early as 700 B.C.
Thales (Miletus)	About 620 B.C. The original principle of all things is water.
Anaximander (Miletus) . .	611 B.C. Conceived an infinite causal series in space and time and the fusion of man, animal, and vegetable and inorganic matter into one cosmos.
Anaximenes (Miletus) . .	About 500 B.C. Air is the primary form of matter, whence all things are formed by compression.
Heraclitus (Ephesus)	About 500 B.C. Fire is the primordial element. Everything is in a state of flux, becoming or ceasing to be.
Anaxagoras . .	500 B.C. Explained physical phenomena as the result of natural causes. One of the earliest accounts of causal sequence, matter consists of atoms organized by divine intelligence (nous).
Pythagoras (Samos) . .	582–500 B.C. Gave an importance to numbers incomprehensible to us, but which probably expressed an idea of harmony and universal law necessary to a cosmos. A vision of immortality. To him is attributed the notion of the four elements earth, air, fire and water.
The Schools of Cos and Cnidus.	Hippocrates of Cos 459 B.C. Praxagoras of Cos. Chrysippus of Cnidus. (The teachers of Herophilus and Erisastratus.)

Also of Ionian race or tradition were Ascelepiades, Themison, Dioscorides, Galen, Oribasius, Alexander of Tralles and many others.

TABLE II.

Alexandria.

331 B.C. to A.D. 641.

After Ionia and Magna Græcia (the Greek Settlements of Southern Italy and Sicily) were finally liberated by Athens, the Golden Age of Athens commenced. Athens remained supreme in philosophy for centuries. But Alexandria led the world in mathematics and science soon after its foundation in 331 B.C. by Alexander.

The Library of Alexandria.	Burned by Julius Caesar's soldiers. Pillaged and burned A.D. 390 by the Christians. Burned, it is said, by the Arabs in A.D. 641.
Herophilus } 300 B.C. Erasistratus }	Founded the medical school at Alexandria. Notable advances in anatomy and physiology.
Euclid, 300 B.C. . .	Settled in Alexandria: birthplace unknown.
Appolonius of Perga, 250 B.C.	Geometry—first treatment of conic sections.
Diophantus, A.D. 275 . .	Algebra—the first treatise on algebra.
Ptolemy, A.D. 139 to 161	The only available authority on ancient astronomy. The ptolemaic system was copied by the Arabs and handed down to the sixteenth and seventeenth centuries A.D.
Philo, about 30 B.C. . .	Famous Jewish philosopher.
Origen, A.D. 185 . .	Immortal Christian philosopher and teacher.
Saint Clement, A.D. 150	The teacher of Origen.
Athanasius, A.D. 296	The hero of the council of Nicæa and the leader in the fight against Arianism.
Students at Alexandria:	
Asclepiades	"Nature would as soon kill as cure a man."
Dioscorides	Contemporary of Pliny. First mention of some mineral drugs.
Soranus, about A.D. 100	Born at Ephesus, practised in Alexandria and Rome.
Rufus, about A.D. 100	First description of bubonic plague.
Galen, A.D. 138 to 201	Relying on Hippocrates and Aristotle, constructed a dogmatic system of medicine which held the field for over a thousand years.
Paul of Ægina, seventh century	The last great Byzantine physician.

very happy time for the Ionians. There were wholesale migrations. Alexander founded Alexandria in 331 B.C., and thereafter Greek scholars mingled very freely with Jews and Egyptians. Alexandria was visited by all who travelled and studied. The famous library must have been in full swing by 300 B.C. In 146 B.C. the Romans conquered Greece and burned Corinth, and again many Greeks migrated to Egypt and Italy. Early in our era the centre of gravity shifted to Rome, for although physicians and scholars travelled freely and studied in many centres, they came to Rome to practise and to achieve fame.

Galen is the most famous physician of this Roman period. Born at Pergamus in A.D. 131, he studied at Smyrna, Corinth and Alexandria. In his practice he followed Hippocrates closely and expounded and developed his teaching. He performed numerous thorough and careful experiments on and dissections of animals, but

TABLE III.

Roman Medicine.

A.D. 100 to 200.

Asclepiades	124 B.C. Born in Bithynia.	Practised with great success in Rome. None of his writings have survived. Regarded as a quack by Pliny. Quoted by Celsus and Galen.
Varro ..	117 to 27 B.C.	Encyclopædist.
The Methodist Soranus.	Last century B.C. Born at Ephesus about A.D. 100	Soranus showed, for his day, great advances in obstetrics and gynæcology.
Lucretius ..	95 to 55 B.C.	"*De Natura Rerum.*" The most famous of the Epicureans. The mystery of life. Materialism and free will. Causality.
Celsus ..	About A.D. 50	Encyclopædist and great classical writer. Responsible for much of the Latin nomenclature used today. His writings are a mine of information about Greek and Egyptian medicine. His influence was not great before the Renaissance.
Pliny ..	Born A.D. 23.	Wrote many books, of which his "*Historia Naturalis*" alone survives. It includes a full account of traditions and practices in the medical practice of his day.
Galen ..	A.D. 130 to 201. Born at Pergamus. Studied at Smyrna, Corinth and Alexandria.	A great physician and brilliant writer. Fully recognized in his own day. Experimented with animals. His teleological bias harmonized with Christianity and won him a place in the "Mediæval Synthesis". He dominated medical thought for more than a thousand years.

he applied his results recklessly and uncritically to the human body. A good example of experimentation is his demonstration of the excretion of urine by the kidney and its passage down to the bladder. Curiously enough, it was not definitely known at this time that urine was excreted by the kidneys. There were wild theories of condensation of vapours and their percolation into the bladder. Galen demonstrated the passage of urine down the ureter and clearly showed the valve-like action of the ureteric orifice. He produced hydronephrosis and hydroureter by ligation of the ureter. He gathered up all his data in the best inductive fashion. But in spite of his great knowledge, Galen had no notion of his own colossal ignorance and no conception of the vast fields of knowledge that still remained to be explored. He had an answer to every question. He had no doubts whatever. This happy know-all frame of mind sprang from his teleological attitude. Everything is to be explained by a "this is for that" proposition. The body is the product of perfect designing—so much so that it is difficult to see how disease is to be accounted for at all. Here is an example:

> The shape of the inner cavities, and the like, have all been determined by a faculty which we call the shaping or formative faculty; this faculty we also state to be artistic—nay, the best and highest art—doing everything for some purpose, so that there is nothing ineffective or superfluous or capable of being better disposed.

Such admiration for the perfection of Nature finds no echo in modern science, which sees clearly the element of contingency in the history of the body and even of the cell. Nature, we say, makes many mistakes and just buries them. Galen was influenced by Aristotle, and for him every organ and tissue was moved by its entelechy as though by a primitive spirit with a will and purpose of its own, omnipotent within a certain scope. He could thus sidestep many problems of mechanism and pretend to despise metaphysics, though he makes the admission that "when we are ignorant of the true cause which is in operation we call it a faculty". He took from Hippocrates the elements, qualities, humours and temperaments, and to these he added an indefinite number of faculties. With

TABLE IV.

The Christian Synthesis.

During the earlier part of the Roman and Byzantine periods, Christians disagreed a great deal among themselves on doctrinal questions. Later, during the fourth and fifth centuries, there was more agreement and doctrine was more or less finalized. But during all the time there was so much preoccupation with the philosophy of religion that technical advances were impeded. This, and the terrible confusion of wars and plagues, are the outstanding features in the background against which medical practice is to be seen.

The First Century ..	The Gospels were written and the Apostolic Creed developed. These emphasized history—the birth, life, death and resurrection of Christ.
323. Accession and conversion of Constantine.	Christianity is tolerated and becomes the religion of the State. There had been persecutions under about fourteen of the emperors, especially Nero, Severus, Decius and Diocletian.
The Œcumenical Councils	
325. Nicæa	The Nicene Creed. The victory of Athanasius and the official end of Arianism. The Godhead of Christ is emphasized.
381. Constantinople	Emphasized the manhood of Christ. Christ was perfect man.
431. Ephesus ..	Emphasized the personality of Christ. Christ was one person, not two.
451. Chalcedon ..	The twofold nature of Christ. Christ had two natures, human and divine.
381. Under Theodosius	The Catholic Church was constituted, after which Catholics were regarded as those who were in communion with the bishops of Rome and Alexandria.
Justinian: Accession, A.D. 527. The Code, A.D. 529. And a later synthesis, A.D. 533.	When Justinian came to the throne, he set about codifying the principal imperial statutes in force. The Code was published in 529. Next, the writings of jurists and commentators were harmonized and published in 533; they completed a synthesis in which doctrine and Church were recognized and integrated with civil and imperial law and custom.

these mental instruments he could explain everything. Thus, the stomach has a faculty of attraction by which it attracts food to itself; a faculty of alteration by which it alters food according to its needs and a faculty of repulsion by which it passes on what it does not need. Nevertheless, Galen observed peristalsis and accounted for it much as we do as the work of longitudinal and circular fibres of muscle. He had no understanding of the circulation of the blood. The liver was the central organ of the circulation and the movement of the blood was a to-and-fro movement in the veins. The arteries contained air and the spurting of blood from the wounded artery was explained by saying that the air rushed out of the arteries creating a vacuum. The blood then flowed into the arteries from pores in the veins and escaped through the wound. It is amazing that the Galenical faculties passed for explanations, and that not for many centuries did physicians drop to the trick of explaining processes by simply giving them names which did nothing more than denote the processes. Of course, it would have been dangerous to criticise Galen. In his own day he could take care of himself. He was a vicious, backbiting critic who would go to any lengths in abuse of those whom he disliked. After his death, his influence continued through his vigorous writings. Later, his works became almost canonical. To have disagreed with him would have been a dangerous heresy.

Although there was no very great advance on Hippocrates in the Roman era, there were some notable features of Roman medicine that were characteristic of Roman orderliness and capacity for administration. Thus Rome provided for her citizens one of the best, if not the best, and most abundant water supplies ever furnished to a great city. The purity of the Tiber was guarded by a curator appointed for the purpose. There were supervisors of the sewers. There was hygienic control of the markets. There were marvellous baths capable of accommodating many thousands of people at one time. There was free medicine for the poor and there may have been even a pharmacopœia, thanks to Celsus. Medical practitioners were constituted as a class with recognized duties and privileges.

TABLE V.

Some of the Great Plagues.

More death, desolation and despair have been caused by epidemic disease than by war. If man has so far failed to prevent war, he has at least made tremendous progress in the control of plagues. History seems to tell us that war is tolerable; but that on the other hand, plagues have been more than the bravest communities could bear.

The Plague of Athens, 430 B.C.	Described by Thucydides, who suffered and recovered. Athens was being besieged on the land side. The whole community was completely demoralized, so that even the most sacred religious rites were neglected. Nevertheless those who recovered tended the sick and carried on the defence.
Syracuse, 396 B.C. ..	The Carthaginian army besieging Syracuse. Described by Diodorus in much the same terms as Thucydides described the plague of Athens.
Five plagues in Rome:	
A.D. 79	Following the great eruption of Vesuvius.
A.D. 125	The "Plague of Orosius". Devastated North Africa.
164 to 180. ? Typhus.	The "Plague of Galen". Described by Galen.
251 to 266. ? Smallpox	The "Plague of Cyprian". Worst in Alexandria.
312. ? Smallpox.	
The plague of Justinian	A.D. 513. Eruption of Vesuvius. A.D. 526. Great Earthquake at Antioch. Plague followed. Described by Procopius. The plague was very severe in Constantinople in 542. It decimated the Eastern Roman Empire and may well have been the principal cause of the fall of Byzantine civilization.
The Middle Ages (bubonic plague).	The four principal Crusades were affected by numerous epidemics. The terrible "Black Death" ravaged the whole world in the fourteenth century. (Described by Guy de Chauliac and Boccaccio.) The first "quarantine" was practised at Venice in 1430. There were numerous other epidemics—influenza, leprosy and scurvy *et cetera*.

Syphilis assumed epidemic proportions in the sixteenth century. The French wished to call it "Neapolitan Disease", but the Italians succeeded in having it called "French Disease" throughout Europe.

All British children have read of the "Black Plague" in London in 1665. Many of us remember that typhoid fever caused more casualties in the South African war than did wounds or other disease. Dysentery decimated the British forces on Gallipoli in 1915 and may have lost that campaign to the British.

The late Roman and Byzantine periods saw a decline in the quality of medical practice. There were several reasons for this. In the first place, the tremendous reputation of Galen established his teaching very early. As Harvey Graham says: "Galen touched nothing that he did not ornament with his learning and injure with his theories." His theories could be and were learned by the dullest of people who had no hope of imitating his example of hard work, exact observation and experiment. Unfortunately his theory rather than his practical example was what most impressed those who followed him. There were other factors which hindered progress at this stage. As in the Greek age, theory and practice tended to be divorced. Practice was not fitting for a man of good birth, and accordingly the scientist and philosopher had no technique and could get for themselves no data. This lack of technique is certainly one of the important causes that limit progress. Thirdly, there were, during the first three centuries of the Christian era, at least five terrible plagues, each of which lasted for years and caused fearful confusion and dismay. The physicians, whether Galens, quacks or magicians, were powerless, and the terror and pessimism of these times present an experience which we can hardly even imagine today. In their despair more and more turned to Christianity, not always out of love for the Christian life, but in hope of benefit from Christian religious medicine in which prayer, the laying on of hands and unction with holy oils were common methods, and those to which the faithful should have prior claims. As Thucydides said of the Athenians in the great plague of Athens: "They saw how sudden was the change of fortune in the case both of those who were prosperous and suddenly died and of those who before had nothing but in a moment were in the possession of the properties of others." Those who were capable of religious feeling turned to Christianity and its offer of a better world. In the absence of religious feeling there was no fear of the laws of God or man. Piety and impiety for these came to the same thing.

These fearful epidemics ravaged the whole ancient world and were very severe in Rome in spite of the advanced ideas of cleanliness and hygiene that were part of the Roman life. At a later date in the Middle Ages and especially in the fourteenth century, the plague caused even greater havoc and despair. Men felt that somehow they could control their own quarrelling and that in these matters right and justice had some chance. But plague was not to be understood. Even Christians admitted that charity was of no avail. There was nothing to do but abandon everything and flee. Nevertheless there were many who stood their ground. The stoic emperor Marcus Aurelius is said to have recalled Galen to Rome during the "Plague of Galen" as the great plague of 164 to 180 came to be called. There is no doubt that the great plagues of these times favoured the growth of Christian dogmatic medicine with its promise of miracles, and discredited technical medicine which could promise nothing. Here is one example of the limiting effect on thought and feeling of an over-all ideology. There are many other examples. Indeed, in every age thought and feeling are conditioned by social, political and religious ruling ideas which limit and direct advances. Nevertheless Christianity, which has so often had a retarding influence on science, has always provided the strongest possible stimulus to the practice of medicine. Christian teaching ennobled the vocations of medicine and nursing and preserved the dignity and significance of the sick and the physically weak, and it liberally furnished the means of caring for them. The Greek idea of beauty as symmetry and elegance of form, typified by Plato's teaching, represents sickness, deformity and weakness as entirely despicable. It might be admitted by the Greek that physical faults were misfortunes; but they were none the less reprehensible. The sick and the weak might therefore be neglected and even vilified. This was not the Hippocratic spirit, but in this regard Plato was more Greek than was Hippocrates. Christian teaching and example were a much-needed corrective to such a cruel and disastrous attitude. How important Christian influence was in this respect in the early centuries of our era may be gauged by the fact that Plato dominated philosophy up to the Middle Ages directly through his own writings as well as through the neo-platonists.

After the Byzantine period the western world slid into the chaos of the dark ages, during which the growing power of the Bishop of Rome was one of the few unifying influences, which, though it did not favour progress in science, was set against the confusion of the seventh and eighth centuries. It is during this period that Arabian medicine comes to the front. It is generally conceded that the Arab contributed little that was original. Nevertheless he performed a great service to the world in holding on to what had been gained in earlier times. The Arab was an inquisitive, interested, tolerant fellow, a great copyist and commentator. He was a reasonably good teacher as well. And he added something to chemical technique, being familiar with the processes of calcination, sublimation and distillation.

It is difficult to say how we should have fared without the Arab. As it all happened, the Arabs were responsible

for the reintroduction of Greek learning to the west. The famous medical school of Salerno was founded about A.D. 800 and flourished for about seven centuries. Although clerics were there both as students and as teachers, it was a lay school not unlike a modern medical school, and it was a meeting place for Christians, Jews and Arabs. Physicians were trained and exported to many countries. We hear of them abroad in the tenth century. In all this the Jew and Arab collaborated freely. They had much in common. Neither was bound by Christian dogma, though each had a religious *a priori*. The Jew was generally superior in intellect and morale, so that some of the praise that is given to the Arab in this phase rightly belongs to the Jew.

In A.D. 800 Charlemagne was crowned emperor of all Christendom. Under his reign Christianity was spread by conquest in much the same way as Mohammedanism was spread by the conquests of Mahomet. At the same time, the famous Caliph Haroun Al Raschid ruled in Bagdad. The Caliph was tolerant of Christianity and allowed Christians to visit the Holy Land. In this way, some Arabian medical knowledge was brought back to the west. Two centuries later, the easy-going Arab was displaced from Palestine by the hostile and intolerant Turk.

TABLE VI.
Arabian Medicine.

1. The Persians, Arabians and Jews were familiar with Greek medicine.
2. The Nestorians, who had schools at Edessa and Nisibis, fled east as a result of persecution in A.D. 489.
3. Nestorians and Jews translated Greek works into Syriac.
4. In A.D. 529 Justinian banished most non-Christian philosophers from Athens and Alexandria, and many went east to Persia.

The Arabian conquests	Damascus 635. Cæsarea 640. Alexandria 642.
Haroun al Raschid ..	763 to 809. During his reign diligent searches for writings of physicians and philosophers were made.
Mesue Senior	777 to 857 } Two Christian translators into Arabic,
Hunayn (Johannitius)	809 to 973 } probably the most active.
The Fihrist (Index of Sciences).	A.D. 987. This index disclosed that many works of Hippocrates and Galen had been translated into Arabic.
Rhazes	Bagdad, 865 to 925. Animal gut in sutures. A prolific writer.
Avicenna	Persia, 980 to 1037. Synthesis of Aristotle, Galen and Hippocrates.
Averroes	Cordova (Spain), 1126 to 1198. Introduced Aristotle to the Christian scholastics.

Bagdad was taken in 1055, Asia Minor in 1071, Jerusalem in 1078 and Antioch in 1084. Turks and Mongols were then in possession of the holy places. The crusades followed soon after. The first crusade was preached by Pope Urban II. By 1099 Jerusalem had been retaken. A second crusade became necessary because of Saracen successes; but no great gain followed this crusade. In 1187 Saladin succeeded in consolidating Saracen power and captured Jerusalem. The third crusade followed, and although this expedition, in which Richard I of England took a leading part, had some success, Jerusalem was not retaken. The fourth crusade was a rather sad and discreditable story. There were several other crusades, and though these were less vigorous they helped to establish outposts of the Latin empire in the east and facilitated commerce and the exchange of ideas. The influence of the crusades on medicine was both good and bad. On the one hand, the first primitive hospitals were built and served by the monks. On the other hand, epidemic disease spread widely through Europe. Epidemics were catastrophes which physicians of those times were powerless to control. There was no understanding of the nature of the contagion, although Avicenna (980 to 1037) noticed the swarms of sick and dying rats that accompanied the outbreak of some of the plagues. Secondly, two edicts were promulgated at this time which had the effect, presumably never intended, of limiting both surgery and the study of anatomy. The first (1163) began thus: "*Ecclesia abhorret a sanguine*"; it was interpreted as a ban on surgery. Probably the Church feared that its members would be too often the unwilling cause of death. Surgery was a dire risk in those days. The second edict (1300) forbade the dismemberment of the dead human body, and was intended to prevent crusaders from macerating the bodies of their fallen comrades in order to bring home their bones for burial. However, this edict did, in fact, prevent dissection of the human body.

At the beginning of the thirteenth century there was a prolific crop of heresies. Whether or not these were due to ideas which had been brought home by crusaders is not very important. The soil was suitable for the growth of heresy. One in particular, a kind of Mannichæism, was considered dangerous enough to justify a crusade on the home front. This heresy was especially prevalent in the south of France and north of Italy. The centre in France was Albi, and the sect came to be known as Albigeois. In Italy the Cathari preached similar doctrines. The essence of their doctrine was the existence of primary and fundamental evil as well as good. Sacraments and churches were reviled and authority was rejected. Suicide was permitted. The prevalence of such doctrines seems to be an indication of the background of pessimism which resulted from the confusion due to wars, dirt, vermin and the widespread misery caused by plagues. In the first day of Boccaccio's Decameron the author gives a word picture of the misery and disorder in Florence. There were some who ate, drank and were merry, expecting death on the morrow. Of the others he writes as follows:

> Tedious were it to relate how citizen avoided citizen, how among neighbours was scarce found any that shewed fellow feeling for another, how kinsfolk held aloof, and never met, or but rarely; enough that this sore affliction entered so deep into the minds of men and women, that in the horror thereof brother was forsaken by brother . . . and oftentimes husband by wife; nay, what is more fathers and mothers were found to abandon their children, untended, unvisited, to their fate, as if they had been strangers.

This was a cruel age. The alarm caused by spreading heresies brought about the Inquisition and was the excuse for savage persecution of the Jews. There had been a burning as early as 385, when a Spaniard, Priscillian, was denounced by the bishops and burned by the emperor. Saint Ambrose and Saint Martin condemned the part taken by the bishops in this event. It was not until the Cathari became so dangerous with their pessimistic and anarchistic notions that heretics came to be punished by death, especially by burning. The horror of heresy and the superstitious dread of witchcraft and sorcery seemed to be linked in the minds of the people. So presently, witches and sorcerers were being ferreted out and burned. Only Roger Bacon, in the thirteenth century, seemed to believe that there were no witches, maintaining that the practice of witchcraft was partly fraud and partly illusion. Except possibly in Spain, the Inquisitors were never so cruel as the mob, and at times the Church had recourse to strong measures to control the mass hysteria and sadism of the crowd. Many of the clergy protested, and in 1076 Pope Gregory VII actually excommunicated the city of Cambrai.

The Middle Ages witnessed the foundation of the universities. Salerno had been a medical school since early in the ninth century. Paris, Montpelier and Bologna had medical faculties early in the thirteenth century. Not all universities can be said to have been "founded". The University of Paris grew like Topsy. The first documentary recognition of the University of Paris was a charter granted by Philip Augustus. It was a concession to students, and like so many similar "privileges", it was given as a solace after the students had been well beaten in a tavern brawl, savagely clawed by the townsmen led by their provost. Thereafter the provost was bound by oath to respect students' privileges, and was appointed "conservator" of the royal privileges of the university. The subjects taught were scholastic philosophy and theology. Law was also studied. This was a scientific development of canon law. So the universities tended to side with the canonists in the lively contest which developed between them and civilians in the thirteenth century (and especially in England), each side claiming

for itself as much jurisdiction as possible. The people generally and the universities favoured the canonists, for it was better to get off with a penance than to be summarily hanged.

In the Middle Ages many of the great cathedrals were built. The light pointed gothic arch made it possible to build high. The East had little influence on European architecture. Side by side with the cathedrals there were great developments in the schools, and presently the cathedral schools eclipsed the monastic schools. Education was less organized than it is today, but was continued longer. The mediæval equivalent of our six to eight years of secondary school and university was as much as ten to fourteen years.

One of the most interesting features of the background of mediæval science is the scholastic philosophy which culminated in the famous treatises of Saint Thomas Aquinas (1225 to 1274). Hitherto Plato, among the ancients, had held the field in philosophy, both directly and through the neoplatonists Plotinus and Porphyry and through the writings of Clement of Alexandria, Origen and Saint Augustine. Since about the fifth century the knowledge of Greek had declined. The incomparable prose of Cicero had helped to make Latin the universal language, and Greek was neglected. After Boethius (450 to 525) no Roman scholars of note had a competent knowledge of Greek science, and Aristotle was known only through the translations of Boethius. Aquinas, however, had direct access to Aristotle's work. More than a century earlier Abelard had fascinated theologians with the logic of Aristotle, and had infected them with the belief that the magic of the syllogism might be used to solve all the riddles of the universe. "A doctrine is not to be believed", he is reported to have said, "because God has said it, but because we are convinced by reason that it is so." Aquinas, however, unlike his predecessors, had a really competent knowledge of Aristotle, and he set about the task of harmonizing the science and the metaphysics of Aristotle with Christianity. In Paris, about 1270, the Dominicans were in some disfavour with the authorities because of their apparent sympathy with the philosophy of Averroes (1126 to 1198). Averroes, who lived in Spain a century earlier, had expounded Aristotle with an eye to the religion of Mahomet. From Aristotle's work he had arrived at a kind of pantheism. The soul, he had said, is individual and not immortal. Only intellect is immortal and intellect is not individual. It is the same under all conditions and in different individuals. According to Bertrand Russell, Averroes is here more in line with Aristotle than is Aquinas. However this may be, Aquinas stuck so closely to Aristotle that when his work became a standard of reference the word of Aristotle also became almost canonical. Presently the work of Galen was adopted and the mediæval synthesis was complete, consisting of the early Christian synthesis, the science and especially the metaphysics of Aristotle and the medicine of Galen. In the background was the idea of the transcendental Rome of the Empire and the Papacy, which grew, it is suggested, from the mystic Rome of Virgil and Saint Augustine.

Modern philosophers do not share the enthusiasm of Abelard and the schoolmen for Aristotelian logic. It is clear now that, even within its limited scope, it harboured errors. It is clear, too, that deductive reasoning was over-estimated, for it does not, strictly speaking, give new facts, though it may indicate how new experiences and new facts may be got. The appearance of unity and finality which Thomas Aquinas gave to mediæval philosophy did not go deep enough. There were differences between the teaching of Dominicans such as Aquinas on the one hand and that of Franciscans such as Duns Scotus, William of Occam and Roger Bacon on the other hand. As far as science was concerned, there was little hope of progress in a world dominated by scholastic philosophy. It was even dangerous to search for new facts and experiences. It was necessary only to know what Aristotle or Galen had said, and the rest was to be got by armchair deduction. Positivist science was needed to make the world more comfortable. Presently the mediæval world broke up in moral and social disorder and schism. There could be no progress in medicine in this phase. Nevertheless, in the midst of confusion the germ of free thought began to spread and prepared the way for later gains. We should here remember the troubadors, those roaming minstrels and lyric poets who sang of feminine beauty and masculine valour. The troubadors became numerous in the eleventh, twelfth and thirteenth centuries in southern France, north-eastern Spain and northern Italy. They sang in their native language, and no doubt they influenced Dante, Petrarch and Boccaccio, who all fell madly in love and told the world about it in their own graceful native language. From the troubadors and the fourteenth century poets we seem to have built up the modern view of marriage as a venture which should be preceded by the boy and girl's falling in love. It was a new idea of freedom that a man should marry for love and enjoy his love of woman.

The Italian Renaissance introduced an entirely new and warm colour into the background. It was, in effect, a Greek era, full of humanism. No longer is the world merely a place of preparation for another existence. Man is henceforth to have the privilege of enjoying this life for its own sake. The accent is at first on beauty, and it is realized that beauty is to be found in the natural sciences. Botany becomes a subject of intense interest. The beauty of the human body which the Greeks knew so well is revealed anew in the works of the great painters and sculptors of the fifteenth century. So the human body becomes an object of direct study. The ban on human dissections is ignored and a renaissance of human anatomy occurs. It is a strange circumstance that the renewed study of anatomy should have resulted very largely from the curiosity of an artist and engineer rather than from the necessities of medical practice. Great anatomists were to appear in the ranks of the medical profession in the sixteenth century; but it was the genius of Leonardo da Vinci (1452 to 1519) which brought the first fruits of the freedom which this age was to develop. What motive can have impelled this great mind to such a minute study of the structure of the human body as is evidenced in his writings and wonderful sketches? We have just noticed that the spirit of this age was a great yearning for beauty, a beauty that could be appreciated by the senses. It is possible therefore that it was the artist in da Vinci which first drove him to the dissection of the human body. The study of anatomy is part of the artist's life. But Leonardo da Vinci examined minutely the whole body, including the brain and the cerebral nerves. He studied also the embryos of animals. His technique included injections of veins, injections of cavities with wax and many gross serial sections; and happily for us he recorded his work in numerous volumes and hundreds of drawings. However, the Renaissance and the Reformation did not bring more than a limited freedom to the sixteenth and seventeenth centuries. In 1553 Michel Servetus, theologian and anatomist, was accused by the Calvinists of heresy, arrested at Geneva and burned at the stake. Servetus had described the pulmonary circulation and thus dealt a blow at Galenism. He was not indicted for this, but for his criticism of the doctrine of the Trinity. But there is no doubt that his opposition to the established ideas of Galen added to his unpopularity. Perhaps the most significant omen of the dawn of an age when thought and reason could be freed from the shackles of tradition and arbitrary authority was the success of the Cartesian philosophy. Descartes (born 1596) published his main philosophical works between 1637 and 1644. He began by doubting everything except the certain fact of his own existence as a thinking being. From this datum only he endeavoured to build up a philosophy which should be free of arbitrary assumptions. After Descartes came Leibniz in Hanover and Locke in England. These philosophers all arrived at a more or less orthodox view of religion by methods purporting to be rational and not bound by the tenets of faith. In the middle of the eighteenth century, however, Hume (1711 to 1776) published his various historical and philosophical works. Hume was the complete sceptic, doubting the validity of all knowledge. In 1776 Edward

Gibbon published the first part of the "Decline and Fall of the Roman Empire", which included a not very sympathetic account of the growth and development of the Christian religion, which is interpreted to mean that Christianity had succeeded not because it was a revealed religion, but merely as the result of human causes. Gibbon was heavily bombarded by the ecclesiastics; but England under George I and Walpole had become very latitudinarian. Gibbon in England and Voltaire and d'Alembert in France were deists. The influence of these and others was linked with the movement which came to be called the Enlightenment. According to deism the universe may be attributed to a very remote God, who set it going and provided it with self-authenticating immutable laws. It did not take the logicians long to declare that self-authenticating laws are such as exist by necessity and need not be attributed to a creator. There is therefore (according to this view) no practical difference between deism and atheism. Whatever may be the faults or merits of the Enlightenment, the toleration of the new ideas free of any body of dogma gave to the scientist a freedom which he had never before enjoyed. If the universe is subject only to immutable laws and is not subject to interference, one has only to discover those laws. Authority which relies on divine revelation need no longer terrify the philosopher, scientist and physician, for it is after all only fundamental law that can be revealed. It is, however, a shallow view to regard the eighteenth century as a time of ebb in religion. In Germany the enlightenment was linked with a strong surge of religious feeling. In England Methodism arose and flourished. It was rather orthodoxy in religion than religion that was neglected. Yet from Joseph Butler within the ranks of the orthodox came one of the most important contributions ever made to moral science.

It was early in the seventeenth century that Francis Bacon had published his treatises on the advancement of learning and the advancement of the sciences. Here we find the first explicit account of the inductive method, which implies that new knowledge is the fruit of experience and is not derived from authority. Though Bacon had little influence in his own day (he was himself no scientist and was unaware of the works of Harvey and Kepler), he had a great influence in more recent times. The scientist now proceeds inductively of set purpose. Inductions presuppose the laborious collection of data which require continuous advances in technique. It is not a matter of surprise therefore that factual details now far outstrip our capacity for induction. This predicament is obviated to a great extent by the statistical method of Francis Galton (1822 to 1911). The idea of the statistical method is inherent in the work of a number of physicians who in the nineteenth century contributed much to the knowledge of epidemiology—and that without the aid of the science of bacteriology. They (Snow, Farr, Budd, Panum and Pettenkofer) had in fact no more data than could have been available to the Galenists or even to Hippocrates. What could have withheld this simple elementary knowledge from the ancients? How came it that the famous Hippocratic method of accurate observation, accurate description and recording gave no clue for two thousand years? It seems that the over-valuation of deductive reasoning has been the limiting factor. It is hard to understand and it may well be that the inductive process is largely an unconscious one. Inductions like the other integrations studied by Sherrington, Pavlov and others have a certain givenness about them and a bias towards armchair deduction has an inhibitory influence. It presupposes a fixed *à priori* which excludes novelty and the new idea. There has thus been a deficient mental technique as well as a deficient manual and instrumental technique, which we are only now in a position to correct.

In addition to the new light which statistical studies have shed on human affairs, it is worth noticing that the eighteenth and nineteenth centuries witnessed numerous simplifying and unifying concepts in the biological sciences —for example: the tissue theory of Bichat; the cellular pathology of Virchow; Schultze's concept of the cell; the comparative anatomy of Cuvier and Vicq D'Azyr; evolution and the work of Mendel and others on heredity. The modern medicine man is therefore a very fortunate fellow. For him the ancients established his vocation as an honourable and dignified calling. They contributed generously, not the least by exploring so many dead ends.

TABLE VII.

Technique—Photography.

Plato (who liked to be different) flirted with the idea that light came from the eye. Avicenna, too, in A.D. 1000, seemed to think so. Aristotle had no doubt that objects gave out light. The Greeks were the first to recognize optics as a mathematical discipline. The most valued dye of the ancients was purple, got from snails. Aristotle observed that sunlight was necessary to its formation.

Roger Bacon, 1214 to 1294	Described the *camera obscura*; but Aristotle had noticed the image obtained through a small aperture.
Leonardo Da Vinci, 1452 to 1519.	Accurately described the *camera obscura*. Made prints of plants directly on to blackened paper.
Fabricius, 1565	Discovered silver chloride.
Christian Huygens ..	The magic lantern with lens.
Johann Schultze, 1727 ..	Used the darkening of silver salts to "inscribe".
Thomas Wedgwood, 1771 to 1805.	(Son of the famous potter) was the first to print on a sensitive silver plate, but could not fix the image.
Ritter, 1805	Achromatic lens.
Sir John Herschel, 1819 ..	Discovered the fixing action of the hypo-sulphites.
Balard, 1826	Discovered bromine.
Braconnet, 1831	The reducing action of pyrogallic acid.
Niepce and Daguerre, about 1822.	Photography with camera and plate; developing and fixing the image.
The French Government ..	In 1839 purchased the "daguerrotype" and gave it free to the world.
William Fox Talbot, 1844..	Photographic prints from negatives.
Frederick Scott Archer ..	The collodian process. In full use by 1880. Made the photomicrograph and its projection possible.
Adolph Steinhel, 1865 ..	The aplanatic lens.
Clerk Maxwell, 1861 ..	Demonstrated the possibility of the three-colour process.
Abbey and Rudolph, 1890..	The first anastigmat lens (manufactured by Zeiss).
Röntgen, 1895	X-ray photography.
Ernst Mach, 1896 ..	Stereoscopic X-ray photography.

TABLE VIII.

Early Technique—Histology (Staining et cetera).

Staining of Dead Tissues.

1714. Leuwenhoek ..	Used crocus to make muscle opaque.
1758. Reichel	Soaked plant stems in coloured plant extracts.
1770. Hill	Stained plant stems in alcoholic cochineal.
1848. Quekett ..	Used fustic and logwood.
1849 Goeppert ..	Discovered carmine.
to Corti	Nuclear staining.
1856. Hartig	

Injection of Coloured Substances.

1723. Ruysh	Injected blood vessels with tallow, wax and cinnabar for microscopic study.
1740? Lieberkühn ..	Similar studies.
1744. Trembley ..	Fed hydra with coloured substances.

Fixing.

1830. Jacobson ..	Used chromic acid.
1830? Müller	Used potassium bichromate.
1860. Schultze ..	Osmium acid.

Colouring by Phagocytosis.

1778. Von Gleicher ..	Fed ciliates with carmine.
1832 to 1838. Ehrenberg	Fed infusoria with coloured substances.

Histo-chemical Colour Tests.

1807. Link	Used the reaction of iron sulphate with tannin.
1825 to 1829. Raspail	Starch and iodine; sugar and sulphuric acid; xanthoproteic test.
1834. Payen	Mercury protonitrate to colour nitrogenous substances.
1896. Daddi	Sudan III.

Let us always gratefully remember that the great body of knowledge which we have today is very largely a heritage, and that our own achievement has been made possible by the blood and sweat of our forefathers.

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PULMONARY INFARCTION AND ANTICOAGULANT THERAPY.[1]

By A. W. MORROW,
Sydney.

THROMBO-EMBOLIC PHENOMENA occurring in all parts of the vascular tree, but particularly in the heart, lung, brain and peripheral vessels, produce the terminal episode in the lives of more people aged over fifty years than any other cause. Whilst the drama and calamity of post-operative pulmonary embolism or thrombosis have been experienced by all, the condition may occur in many other medical and surgical illnesses. It compels attention because post-mortem studies have revealed that a large number of cases escape clinical detection, and clinical experience has shown, not only that diagnosis and treatment are at times difficult, but that the mortality rate is also very high. However, the doctor is now no longer entirely helpless to avert this disaster.

Modern anticoagulant therapy in pulmonary embolism or thrombosis may be as dramatic as penicillin therapy in pneumococcal pneumonia; but the early clinical recognition of this pathological process is essential for its successful treatment. The diagnosis is unlikely to be missed when there is a complaint of sudden pleural pain with dyspnœa followed by bloody sputum in a patient who has just had an operation or a confinement, or in one suffering from cardiac failure; but there are many other manifestations of this malady which are not recognized so readily, nor is it generally appreciated that it may simulate many other conditions.

Predisposing Factors.

Pulmonary infarction is prone to occur in certain maladies or pathological states,[1] and an awareness of the predisposing factors is essential for the early recognition of the disease. Such predisposing factors are (i) a history of previous thrombo-embolism during illness or the post-operative period, (ii) extensive varicosities, (iii) malignant disease, particularly occurring in the pelvis or abdomen, (iv) extensive abdominal and pelvic operations, (v) fracture of the pelvis and femora, (vi) obesity, (vii) cardiac insufficiency, (viii) advanced age and preceding operation.

Mechanism of Pulmonary Infarction.

It is generally agreed that infarction results in the main from pulmonary embolism; but embolism may occur, even with production of symptoms, without proceeding to infarction. It is not improbable that in some cases pulmonary thrombosis occurs without antecedent embolism and may result in infarction. However, the consensus of opinion favours the embolic origin of infarction, and the source of the embolism is usually found in the deep veins of the calf or foot, or in the pelvic venous plexuses.

It is not unusual for pulmonary episodes to occur before signs of peripheral thrombosis are apparent. This is easily understood when it is realized that phlebothrombosis of the deep veins is a comparatively symptomless event unless diligent search is made for clinical signs. Pains and œdema occur when the process has spread to involve one of the larger veins and the vessel wall has become involved in an inflammatory process—a condition now regarded as thrombo-phlebitis in contradistinction to phlebo-thrombosis. At this stage the clot is firmly fixed to the vessel wall, and the possibility of embolism is distinctly less than in the earlier phase.

Any condition which leads to stagnation or pooling of blood in the venous circulation predisposes the subject to phlebo-thrombosis. Although it occurs predominantly in those conditions mentioned under the heading of predisposing factors, it can occur in ambulatory patients and healthy individuals. Sitting in one position for a long period, such as in an aeroplane journey, has been observed to cause phlebo-thrombosis in one calf which led to pulmonary embolism erroneously diagnosed as coronary infarction. Another patient developed the syndrome after sitting in a tourist omnibus for some hours, during which time she nursed a reasonably heavy attaché case on her knees. In the search for possible sites of peripheral thrombosis as sources of embolism, recently thrombosed hæmorrhoids or prostatitis causing thromboses in the prostatic venous plexus must be considered.[2]

Clinical Syndrome of Pulmonary Embolism and Infarction.

The Classical Syndrome.

The classical syndrome of pulmonary embolism and infarction[3][4] consists in the sudden onset of dyspnœa with pleuritic pain and perhaps blood-stained sputum, with the early development of râles and a friction rub going on to consolidation. Fever is present with leucocytosis and a raised blood sedimentation rate. This syndrome, whilst classical, is frequently replaced by other manifestations.

Pneumonia.

Signs and symptoms consistent with pneumonia[4] are by no means infrequent. The post-operative pneumonia which fails to respond to antibiotics or chemotherapy is almost certainly due to pulmonary embolism or infarction.

Pleurisy.

Pleurisy with effusion which may be hæmorrhagic is not uncommon. This syndrome is not infrequent in cardiac failure.

Coronary Heart Disease.

Angina-like pain or a syndrome almost indistinguishable from coronary infarction may occur.[4] It would be reasonable to state that the majority of so-called post-operative coronary episodes are in fact due to pulmonary emboli or infarcts. In pulmonary embolism the onset is usually sudden and overwhelming, with chest pain which is sharp and without typical localization; whereas in coronary occlusion the onset is more gradual, with prodromal symptoms in the previous twenty-four hours, and pain of definite distribution which may be severe, crushing and constricting, but not sharp. Dyspnœa, cyanosis, shock, rapid heart action and low blood pressure occur very early in pulmonary embolism, whereas in coronary occlusion the signs develop more slowly, and as a rule are not so intense. Fever, leucocytosis and an increased blood sedimentation rate appear early in pulmonary episodes, and may reach conspicuous heights within several hours, whereas in coronary episodes such phenomena do not appear for twenty-four to thirty-six hours.

At times even with the aid of radiography and electrocardiography the differential diagnosis may be difficult.

Acute Heart Failure.

Paroxysmal dyspnœa with pulmonary œdema and cardiac dilatation may occur when a large pulmonary vessel is obstructed. The clinical picture is that of acute cardiac failure.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on December 9, 1948.

Syncopal Attacks and Cerebral Manifestations.

Sudden loss of consciousness, the result of vascular collapse, is much more frequent in pulmonary embolism than in heart disease. Convulsions have been recorded, and in some cases cerebral embolism secondary to pulmonary infarction may be the first indication of the underlying lung disorder.

Disordered Cardiac Action.

Paroxysmal tachycardia, flutter or fibrillation may result from pulmonary embolism, whether the heart is diseased or not prior to the pulmonary episode.

Pyrexia of Unknown Origin.

A number of cases have been reported in which unsuspected episodes of pulmonary embolism or infarction have been responsible for pyrexia of many weeks' duration.

Acute Abdominal Emergencies.

Lung infarction can produce acute upper abdominal pain with rigidity, which, when the cause is unsuspected, has led to exploratory laparotomy.

Sudden Death.

Dramatic sudden death is well known. A careful review of a number of such deaths reveals that in the majority of cases prodromal signs, such as mild pyrexia and even previous minor pulmonary episodes, were present, but often they have been neglected by the attending physician or surgeon.

Ancillary Aids in Diagnosis.

Radiography and electrocardiography are frequently helpful in diagnosis.

Radiography.

Infarcts are often ill-defined or not seen at all during the first twenty-four hours, and thus daily examinations are at times necessary.[2] The lesion is always in contact with a pleural surface, either at the interlobar fissures or at the periphery of the lung. Two or three pleural surfaces are commonly involved by a simple infarct—for example, at the junction of fissures or at the costo-phrenic angles. The medial or cardiac margin of an infarct is convex or "hump"-shaped, and sharp in outline. Oblique and lateral views are often necessary to demonstrate the lesions. Pleural effusions may or may not be present and may obscure the lesion for a few days. Incomplete infarcts may disappear within two or three days, whereas complete infarction persists for two to three weeks and heals by linear scarring. Large infarcts may persist for months with very little change. The site of the infarct is most commonly in the lower lobes and predominantly on the right side. However, other lobes are involved, not infrequently in association with the lower lobes. At times the appearance is that of hazy horizontal streaking at the lung base with some elevation of the diaphragm. This streaking may simulate localized patches of atelectasis. Secondary bronchopneumonia may cause the edges of the shadow to be feathery rather than well defined.

Electrocardiography.

In 1935 McGinn and White[7] called attention to the electrocardiogram in "acute *cor pulmonale*". They described the following significant changes: (i) prominent *S* wave and low origin of *T* wave in Lead I; (ii) a gradual "staircase" ascent of the *S-T* interval from the *S* wave to the *T* wave in Lead II; (iii) conspicuous *Q* waves and inversion of *T* wave in Lead III; (iv) in some cases abnormal direction of *T* wave in Lead IV without alteration of *QRS* complexes. Restoration may occur within forty-eight hours. The appearance may closely resemble that found in posterior wall infarction.

More recently Durant *et alii*[8] have drawn attention to other and even earlier changes in the electrocardiogram. They state that within the first six hours of the incident, changes characterized by intraventricular conduction of the atypical right bundle branch type may be apparent. Within twelve hours normal intraventricular conduction may be reestablished, and then follow the changes described by White. Nevertheless no one electrocardiographic abnormality is consistently present in pulmonary embolism or infarction.

Blood Changes.

A high neutrophile leucocytosis may be present early, together with a greatly accelerated blood sedimentation rate.

Treatment.

Treatment may be summarized as follows.[9]

The immediate treatment consists in adequate oxygen therapy together with the intravenous administration of papaverine hydrochloride (half to one grain) and atropine sulphate (1/100 to 1/50 grain). Anticoagulant treatment must be commenced immediately according to the routine of Cummine. Morphine should be avoided if possible. Heparin appears at times to produce some relief of distress.

The continued treatment consists in repeated injections of atropine and papaverine if necessary, the administration of oxygen as needed and maintenance of anticoagulant therapy. The use of antibiotics or chemotherapy is indicated only when infection becomes superimposed.

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ANTICOAGULANT THERAPY.[1]

By HAROLD CUMMINE, M.D., M.S., F.R.A.C.S.,
Sydney.

THERE is no doubt that in recent years there has been a tremendous revival of interest in the manifestations of intravascular thrombosis, from both a clinical and a pathological viewpoint. The advent of the anticoagulant drugs heparin and dicoumarol into the therapeutic arena have been responsible in large measure for this clinical renaissance. The day has passed when the occurrence of this complication after operation could be regarded as an act of God, and the patient condemned to weeks of immobility with a most guarded prognosis. Early ambulation, control of infection with the antibiotics and sulphonamides and the use of the anticoagulants have made an immense reduction in the morbidity and a miraculous change in the therapy of thrombosis. However, the battle is too recent for the indications for and technique of safe administration of the anticoagulants to have been evaluated properly, and it is my privilege tonight to make some report to you of what has been my personal experience in this matter.

Anticoagulant Therapy.

When thrombosis develops in post-operative cases, either in the legs or in the lungs, the use of anticoagulant

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on December 9, 1948.

therapy is considered to be essential. A series of 158 patients with major pulmonary thrombosis have been treated with four deaths. Also many patients with minor manifestations, which had been diagnosed as pleurisy, atypical pneumonia and pleurodynia, have been treated; no doubt most of the lesions were of thrombotic nature, but owing to the element of doubt which exists in the clinical diagnosis in such cases they are not included for discussion. In none of the 158 cases of major episodes was the diagnosis in doubt. The fatal cases were as follows.

CASE I.—The patient had bilateral thrombosis of the legs and lungs. Anticoagulant therapy was suspended. Sudden death occurred. No autopsy was performed.

CASE II.—The patient had bilateral thrombosis of the legs and lungs following prostatectomy. Progressive congestive cardiac failure developed and death occurred. Dicoumarol was not then available and heparin was scarce, so anticoagulant therapy was wholly inadequate. Thrombosis was evident at autopsy.

CASE III.—Bilateral thrombosis of the legs and an infarction at the base of the left lung followed cholecystectomy. This patient was adequately treated with anticoagulants, but an abscess developed in the infarct and death occurred. The pathological condition was demonstrated at autopsy.

CASE IV.—Thrombosis of the legs and pulmonary infarction followed prostatectomy. Anticoagulant therapy was adequate. The patient became well, but prior to his leaving his bed some days after cessation of heparin therapy, a "cover" dose of 5000 units was given to combat the possible effect of any embolic fragments detached by ambulation. The patient died immediately. At autopsy only the old lung infarction was found, healed.

In Cases I and II death was due to inadequate anticoagulant therapy. Both these cases occurred early in the "feeling our way" period. This lesson has been learned. Case III was quite recent. Death was due to the formation of the lung abscess. This appears to be a lethal complication, and it is believed that penicillin should be used in the routine treatment of all major lung thromboses to prevent such a development. In Case IV death seemed almost certainly to be due to the administration of heparin to a patient who had developed a sensitization to the drug from previous administration. Such a clinical phenomenon had not to my knowledge been described in the literature, and at first doubt prevailed. However, five further (non-fatal) cases have been encountered. In all the symptoms were clear-cut. Whilst the intravenous injection of heparin was being given the patient complained of severe pain in the loins and felt faint. In Case IV, the whole five millilitres (5000 units) were given quickly and the die was cast. The other instances were all observed in cases in which their possibility was anticipated. Whenever heparin has to be given again to a patient to whom it has been administered previously, seven days or more before, the initial dose is given in a graduated manner. The needle is introduced into the vein and 250 units are given, and then a period of five minutes is allowed to elapse. If nothing untoward happens a further 250 units are slowly injected and a further delay of five minutes is observed. This method is continued then with doses of 1000 units until the full quantity has been given. The five patients showed distress during the first ten minutes. In three instances a generalized erythematous rash was present on the following day. The shortest intervening period was seven days and the longest three months.

A series of 325 patients with thrombosis of the deep veins of the legs, either unilateral or bilateral (no lung thrombosis being present), have been treated with anticoagulants. In one instance, whilst the patient was under treatment, complaint was made of a transitory sharp pain in the chest. No clinical or radiological signs were found. When treatment was commenced within six hours of the first report of pain or swelling of the leg, a normal limb was almost always the end result. If twelve hours had elapsed the results were not always so satisfactory in a minority of cases. Treatment instituted later than this gave a higher percentage of poor results. However, it was always given, as it seemed to confer an immunity against a major chest complication and a protection against the involvement of the other limb. One death occurred in this series. After 800 milligrammes of dicoumarol had been given, severe and protracted melena occurred, for the control of which 19 litres of blood and much vitamin *K* were required. The patient recovered from the hæmorrhagic complication and was discharged from hospital, but with a low-grade pyæmic infection of the right costo-clavicular joint and cervical vertebræ. He died shortly after from what appeared to be a cerebral thrombosis. Although death was not directly due to dicoumarol administration, there appeared little doubt that the profound anæmia which developed at least to some degree contributed to the development of the pyæmia.

When a patient with post-operative thrombosis is given anticoagulant treatment, a standard method of administration is advisable. The essential principle is the combined use of both heparin and dicoumarol until the patient is ambulant. If bed rest cannot be avoided, then the anticoagulants must be continued for as long as gross amounts of fibrinogen B are present in the plasma (see later). It has been found from experience that in post-operative and post-partum cases it is usually possible to have the patient up and about by the fifth day of treatment, and because of this it is only in a few cases that the use of anticoagulants for more than five days is required. Heparin is given for the first two days at four-hourly intervals in doses of 5000 units. The first five doses (25,000 units) are given as a routine measure, but during the next twenty-four hours heparin is given only if the four-hourly estimation of the coagulation time falls to below ten minutes. This is usually all the heparin that is required. It is convenient to give the heparin into the tubing of an intravenous drip apparatus for the administration of saline set to run at 30 drops per minute. It may be necessary to reinsert the needle if the intravenous apparatus is used for the second twenty-four-hour period, as blockage of the vein is not uncommon, especially if a sugar solution is used.

The first dose of dicoumarol (300 milligrammes) is given when the heparin treatment is commenced. The next dose (200 milligrammes) is given twenty-four hours later and another of 100 milligrammes on the third day of treatment.

At this stage it is most essential to estimate the prothrombin percentage of the plasma, as a great deal of information is obtained about the future management of the patient. For practical purposes the prothrombin index will fall into one of three groups which are selected arbitrarily.

Group I: Prothrombin Index 100% to 70%.

The first group may be termed the non-responsive group. The first 300 milligrammes were given at least forty-eight hours prior to the test's being performed, and so it can be expected that all this amount should be producing an effect. The second dose of 200 milligrammes was given twenty-four hours before, and may or may not be producing a partial or complete effect. None of the 100 milligrammes given on the day of the test will be effective. As there is no demonstrable reduction of prothrombin activity in this group, either of two states of affairs may be present. The patient may be resistant to dicoumarol or may merely be a "slow starter". Therefore it is essential to reestimate the prothrombin index the next day (fourth day of treatment). If there is still no decrease in the prothrombin index, then in all probability the patient is resistant and the further use of dicoumarol is deemed inadvisable. In such cases, if ambulation cannot be achieved and fibrinogen B is still present, then the coagulation time is estimated by the capillary tube technique at four-hourly intervals, and 5000 units of heparin are given intravenously when the time falls below ten minutes. As soon as the patient is ambulant or the plasma is free of fibrinogen B, this is stopped. From experience it is rare to find patients requiring this method of therapy beyond the sixth day of treatment. Table I illustrates the management of this type of case. However, should estimation of the prothrombin index on the fourth day show a depression, then the patient is a "slow starter" and the subsequent management is as described for Group II.

Table I.

	Day.					
	First.	Second.	Third.	Fourth.	Fifth.	Sixth.
Heparin	5000 units intravenously at four-hourly intervals.	5000 units intravenously to keep coagulation time (estimated four-hourly) above 10 minutes.	5000 units intravenously, to keep the coagulation time (estimated four-hourly) above 10 minutes.			
Dicoumarol	300 milligrammes	200 milligrammes	100 milligrammes			
Prothrombin index			100%–70%	100%–70%	Repeat estimation if patient not ambulant.	

Group II: Prothrombin Index 70% to 50%.

Into the second group fall the greater number of patients. It can be forecast that the prothrombin index will be below 50% up to the fourth and fifth and possibly the sixth day. If ambulation is possible no further anticoagulants are needed, and from experience convalescence is usually uneventful. If the prothrombin index recovers rapidly, a further 200 milligrammes may be necessary on the fifth day if movement is not satisfactory. This provides cover for the seventh and eighth days.

Group III: Prothrombin Index 50% or Less.

In the third group the response to dicoumarol has been too good. The clinician's fears are transferred from thrombosis to the possibility of hæmorrhage. This group is apparently one of hyperreactors. It is essential to carry out daily estimation of the prothrombin index, and should the value fall to below 20%, the necessity for active measures to prevent further depression must be considered. The decision to be made by the clinician is whether to intervene with blood transfusion and vitamin *K* or to leave things alone. The decision must be made, not upon laboratory figures, but upon clinical factors. Firstly, a major hæmorrhage when only 600 milligrammes have been given to an adult is exceedingly rare. Nor are minor hæmorrhages very common. Secondly, many cases are encountered in which the prothrombin index is less than 20% and the progress is uneventful. Thirdly, the whole object of therapy was to depress the prothrombin activity, and measures to restore it rapidly to a higher level may defeat this essential purpose.

Practice in the Present Series.

The practice adopted in the series treated so far has been to intervene if the general condition of the patient was such that the onset of hæmorrhage appeared likely to make the prognosis grave. It was found from practical experience that when the thrombosis involved the leg, intervention was rare, whereas major involvement of the lung invariably required active measures. This viewpoint is a purely personal one and may well be modified by further experience. A most important factor in assessing these cases is the place where the patient is being treated. One would feel far happier to adopt expectant treatment in the better equipped hospitals, especially where the immediate attentions of a resident medical officer were at hand, than in other less ideal situations. The availability of pathological laboratory services may well be a further influencing factor.

Pulmonary Thrombosis—Primary or Embolic?

For some time it has been accepted that almost all lung thromboses are embolic in nature, arising from preexisting thrombosis of the peripheral venous trunks, especially of the legs. This is, of course, a neat mechanical conception which adequately explains some of the cases; but there is a body of embarrassing evidence which cannot be disregarded, and which throws some doubt upon this "universally embolic" theory. In most reported series the first clinical manifestations of the disease appear in the lungs in approximately half the cases. One may, of course, regard the lung condition as due to an embolism from a "silent" phlebothrombosis of the leg. This is, at the most, an hypothesis, as the great number of patients recover and the opportunity for proving the contention is limited. To prove this belief is as hard as to disprove it. Some of the earlier autopsy series reported involvement of the leg veins in up to 90% of cases of pulmonary thrombosis, and it is largely upon these reports that the embolic conception is based. However, in assessing the importance of these reports one must remember that by the time the patients died the process of intravascular thrombosis might have been progressing for days or even weeks, and it would be surprising indeed if clots were not found in the leg veins of most of them. That they are there by no means proves that the lung thrombus arose from them. All autopsy surgeons have experience of cases in which detailed examination of the peripheral vessels reveals neither clot nor "donor site". This cannot lightly be overlooked. Primary lung thrombosis does occur; the question to be decided is not its existence as an entity but its frequency as a clinical phenomenon.

The use of heparin in the treatment of established leg thrombosis throws some light upon the problem. Murray of Toronto reports 371 cases treated with heparin, in which no lung embolisms followed treatment. In a comparable series treated by myself, an identical experience has prevailed. It seems incredible that in nearly 700 cases "embolism" has been prevented by heparin. What seems much more likely is that primary lung thrombosis has been prevented by the use of the anticoagulants. In reverse, experience has been similar. When a patient with lung thrombosis is adequately treated with anticoagulants, it is rare for a thrombosis of the leg to develop. This was not the state of affairs before the advent of heparin and dicoumarol.

Another interesting aspect of the problem is that in reported series in which femoral vein ligation is practised, subsequent "emboli" still occur.

When "pulmonary embolism" was treated by Murray in 149 instances there was no fatality. A personal series of 158 cases with four deaths has been described. Of these fatal cases, one only could be due to embolism and no autopsy was performed. Furthermore, this patient had not been under anticoagulant therapy for some time before death occurred. One patient died from infection superadded upon infarction, another from heparin sensitivity, and the third from progressive cardiac failure following inadequate anticoagulant therapy. It seems reasonable to say, then, from the experience of these two series, that if the patient survives long enough for heparin therapy to be commenced, then death appears unlikely. If the majority of these cases were due to a massive preformed embolus, then it seems difficult to understand how heparin could produce so consistent a "save", as the damage would have been done before therapy was given. There are two possible explanations. A large embolus may strike the patient but not kill him immediately. In a short time, however, local blood clotting may be superadded upon the thrombus in the lung vessel and cause death. The prompt use of heparin would prevent this and explain the results obtained. An alternative explanations may be that a small thrombus has developed silently in the pulmonary

vessel, and then suddenly massive local clotting occurs upon this foreign body. Some find it difficult to accept the idea that spontaneous intravascular thrombosis may occur in an artery. Figure I, which illustrates such a condition in the descending aorta and common iliac vessels, may cause some doubts to arise upon this fixed viewpoint.

Whatever the opening move in the drama, primary thrombus or initial embolus, it does seem quite clear that if the patient survives the initial shock and heparin is given, then death appears unlikely, and that the most probable explanation of this is that the heparin prevents the superadded massive clotting upon the initial focus. This is the essential fact for the clinician; the point and counterpoint of ætiology are for the academician.

One must now consider those patients with sudden lung involvement who perish before any therapy can be instituted. This group is not included in my series here reported or in those of Murray. Are these instances of true sudden massive embolism? No doubt some are, but

FIGURE I.

this may not be so invariably. The histories of 25 such instances were studied carefully in an endeavour to demonstrate the occurrence of some warning phenomenon which preceded the catastrophe. In five cases nothing could be found; an apparently perfectly well patient was struck down in a few minutes. In two of these cases there was some thrombus in the peripheral veins, but no abnormality could be found in two others. No autopsy was performed in the remaining case. In twenty of the cases a premonitory episode was present in retrospect, but its importance had been overlooked. Such episodes were pleurisy, low-grade pyrexia, tachycardia, minor syncope, coronary occlusion, thrombosis of the leg, undiagnosed swelling of the leg on getting up and a feeling of being "off colour" for a day or more. In several instances more than one of these had been present. It may be argued that all this was evidence of an intravascular thrombosis occurring in the peripheral veins, which in some cases actually gave rise to minor emboli before the detachment of a final fatal embolus. This may be so, but it in no way disproves the contention that what finally killed at least some of these people was a massive thrombosis rapidly developing in the pulmonary vessels. In support of this hypothesis one may cite the experience when all such minor episodes occurring in post-operative and post-partum patients are treated with anticoagulants and ambulation. There is an almost complete elimination of subsequent complications. This technique has been practised for almost two years in the male section of the department of urology, Royal Prince Alfred Hospital, and in one instance only has a thrombosis occurred. This was

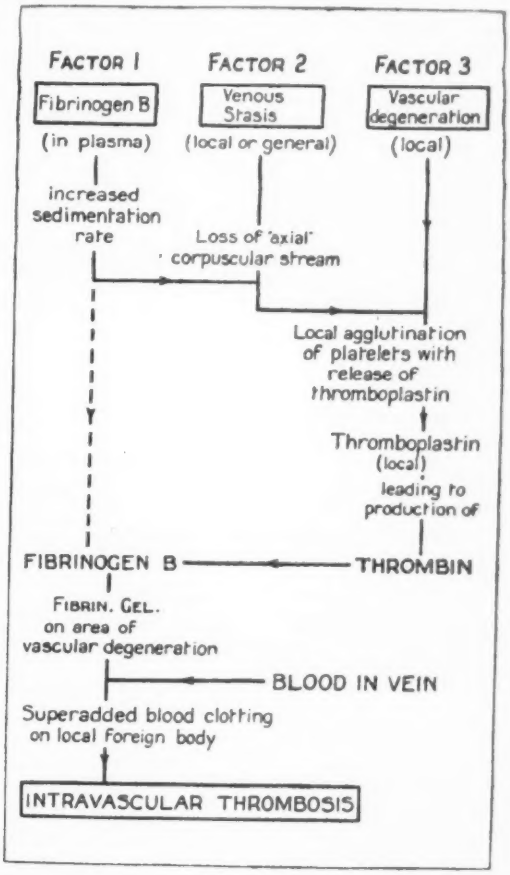

FIGURE II.

in a post-prostatectomy patient and due to a change in ward administration; when the criteria of control were not observed the first case of thrombosis in the two-year period was allowed to occur. The patient had a low-grade pyrexia and complained of pain in the chest. His coagulation times were not estimated and the diagnosis of "pneumonia" was made and held for twenty-four hours. The lung thrombosis progressed until anticoagulant therapy was commenced, and thereafter recovery began.

A parallel experience is being accumulated in the professorial surgical ward. As yet the time interval is too short; but if at the end of a five-year period these findings are consistent, then the belief in a universal embolic origin of pulmonary thrombosis will require drastic revision.

The Role of Fibrinogen B in Thrombosis.

It has been appreciated for some time that after operation and child-birth an increase in the plasma fibrinogen level is usually present. This was regarded as a quanti-

tative increase. However, following the description by R. N. Lyons of a form of fibrinogen (which he termed fibrinogen B) as an intermediate stage between normally occurring plasma fibrinogen and fibrin in the steps of blood coagulation, it may well be shown that this increase of fibrinogen is of a qualitative nature. Should this be proved so, then the occurrence of this intermediate form of fibrinogen in gross amounts in post-operative non-ambulant patients may be a major factor in the onset of intravascular thromboses.

In cooperation with Lyons I have studied this aspect of the problem for almost three years, and the opinion has been formed that the presence of fibrinogen B in the plasma is of prime importance in the pathology of intravascular thrombosis. It is improbable that fibrinogen B is the sole contributing factor in the onset of this condition. Two additional (and equally important) factors are venous stasis, either localized or general, and endothelial changes of the vessel wall of traumatic, infective or degenerative nature (see Figure II).

It has been found that in cases in which little or no fibrinogen B develops after operation, thrombosis does not occur. There are two exceptions to this finding. Localized clots may occur in varicose veins and also in veins where an indwelling cannula has been placed for intravenous therapy. In the first instance the factors of localized venous stasis and gross vascular degeneration are operating, and in the second local trauma to the vessel wall and such physico-chemical irritation as may result from the instilled fluids.

FIGURE III.

Photogram of capillary tube with contained blood. This is the actual size of the tube and gives a satisfactory impression of the bore of the capillary tube and the thickness of the column of blood, which is seen in the middle portion of the tubing. The bore of the capillary tube should never be greater than that illustrated.

There is another form of thrombosis which has been observed to occur in the absence of fibrinogen B. In patients who have developed a thrombosis of the leg or lung and are recovering, one may find that a further thrombosis develops even when repeated examinations have shown the plasma to be free of fibrinogen B. Cases of this type are rare, and I have encountered only eight. On two occasions the leg and on six the lung was involved. The striking point was that in all instances the recurrence was at the site originally affected, and the impression has been formed that this was due to normal blood clotting occurring upon the previous intravascular clot, which acted as an intravascular foreign body. This is not a difficult hypothesis to accept, as the mechanism involved is a normal physiological one. In the lung cases one of course cannot exclude embolism in any given instance, although my personal impression is against this as the explanation of the phenomenon.

The value of fibrinogen B estimations is both prophylactic and prognostic.

Prophylaxis.

If serial fibrinogen B estimations are carried out every three days after operation or child-birth, two states of affairs may be found. There is a small group of patients in which the result of the test is consistently negative. Thrombosis has not been found to occur in these patients except in localized varicose veins or after intravenous therapy. Most patients, however, fall into the group in which fibrinogen B is seen to develop. It is commonly present in the second week. The fact of its presence does not indicate that thrombosis is to be expected. Indeed, on the contrary, it may be the normal state of affairs. When there is no pyrexia and the patient is moving actively in bed or is ambulant and the capillary coagulation graph is of a normal type, then special precautions are not indicated.

If, on the other hand, these criteria are not present, then the prophylactic use of heparin, and ambulation when possible, must be considered. It is my personal practice to use heparin, unless it is otherwise contraindicated, when the following findings are present after operation: (i) confinement to bed (venous stasis); (ii) pyrexia (especially of low-grade intermittent nature); (iii) low blood coagulation times (three to five minute range); (iv) presence of fibrinogen B (in more than slight amounts).

The appearance of signs or symptoms in the legs or lungs, which should be sought for daily, and which may reasonably be considered as thrombotic, renders the use of heparin an immediate "must" rather than a possible "perhaps". However, this is rather on the late side, as thrombosis has begun and the essential object is to prevent it.

Prognosis.

When a thrombus is present either in the lung or in the leg, anticoagulants should be used. There exists some difficulty in deciding for how long their administration should be continued. Most of the disappointments in the use of heparin have arisen from the fact that as soon as treatment is stopped the liability to thrombosis is again present within a few hours and recurrences are not uncommon. With dicoumarol a satisfactory depression of the prothrombin index must be achieved, and this is not without danger. Indeed, many clinicians are doubtful which to fear most—the Scylla of thrombosis or the Charybdis of hæmorrhage. The problem may be solved largely by using the presence of fibrinogen B and ambulation as the two governing prognostic factors. If the patient is able to get up by the fourth day of anticoagulant treatment, and if fibrinogen B is absent from the plasma, then further thrombosis has rarely been seen in my own cases. If the third criterion of a normal coagulation time is added, then the patient appears to be immune from further complication. (See Figure III for approximate size of capillary tube used for estimation of coagulation time.) In those cases in which fibrinogen B persists and confinement to bed is essential, anticoagulant therapy must be continued, as it is in this group that recurrences have been encountered. It is considered essential to keep the coagulation time consistently in the six to ten minute range in these cases. In the treatment of patients who give negative responses to fibrinogen B tests, but cannot leave their beds, it is our practice to persist with anticoagulants to keep the coagulation time up, in order to prevent those locally recurring thromboses which have been described previously, if there is pyrexia or active bed movements are not possible.

After a thrombosis the serial fibrinogen B findings may be of three types: Type I: no fibrinogen B found; Type II: fibrinogen B absent for a day or so, then present again; Type III: fibrinogen B present all the time.

As has been described, when thrombosis occurs a standard course of heparin and dicoumarol is given irrespective of the fibrinogen B findings. This carries on until the third day when the prothrombin index is estimated. From the same specimen a fibrinogen B test is performed. If the result is negative, no further anticoagulants are necessary provided that the patient can leave his bed. If fibrinogen B is present and the patient cannot leave his bed that day, then further administration of anticoagulants in the manner described is indicated. It is considered that a negative fibrinogen B finding should be confirmed next day.

It can be said that when the continued use of anticoagulants is governed by these factors, the cases in which confusion arises are very few, and almost all the uncertainty so characteristic of thrombosis seems to disappear. The amount of drugs used is reduced to a minimum, and where heparin is concerned this is no small item. In the case of dicoumarol the risks of hæmorrhage are reduced to the lowest possible clinical margin.

Summary.

1. A method for administering anticoagulants to give optimum effect with minimal danger is described.
2. Some criticism of the theory of universal pulmonary embolism from the peripheral venous circulation is made.
3. The pathological background of intravascular thrombosis is discussed, with special reference to the form of fibrinogen described by Lyons and termed fibrinogen B.
4. The detailed biochemical evidence for the character and metabolism of fibrinogen B is not dealt with, as this is the special field of Lyons, who is at present abroad, and on his return in the near future it will form the subject of a paper.

PSYCHOSOMATIC ASPECTS OF GENERAL MEDICINE.[1]

By HAROLD R. LOVE, M.B., B.S., M.R.A.C.P.,
Brisbane.

Felix qui potuit rerum cognoscere causas
atque metus omnis et inexorabile fatum
subiecit pedibus strepitumque Acherontis avari.
—VIRGIL.

THE term functional was introduced into medicine to describe a set of disorders in which disturbance of function occurred in the absence of organic disease. To Walter B. Cannon belongs the credit of insisting that a concept of functional variation without structural change is unreal and misleading; and of suggesting that the clinician might escape on the one hand from the demands of the pathologist for physical evidence of organic disease and on the other from the "vagueness and mysticism of the psychological healers" by studying the physiology of strong emotion. Cannon set the lead by his own classic studies on the subject.

The word psychosomatic was coined to express this idea as applied in clinical medicine. It is a bad word since it emphasizes the psychiatric approach to functional disturbance, which Cannon intended that the clinician should avoid. Nowadays it is becoming more and more clear that functional or psychosomatic disorders have a characteristic and complex physiology which must be elucidated if the clinician is to distinguish them accurately from the physiological disturbances due to organic disease, and so treat and prevent them scientifically and efficiently.

The modern accumulation of post-mortem data from subjects who have undergone prefrontal leucotomy, massive sympathectomy and similar procedures, together with the newer anatomical researches of Le Gros Clarke and others, now leaves little doubt that the frontal lobe of the cerebral cortex is almost entirely concerned with visceral and emotional sensation and with motor visceral control. The cortex receives via thalamic, hypothalamic, cerebellar and similar relays all available information as to the states of the external and internal environments of the individual. Through the extrapyramidal, cerebellar and cortico-neuraxial pathways it imposes upon the skeletal muscular system the locomotor, manipulative and phonative patterns of activity which make up physical behaviour. At the same time, through the thalamus, the hypothalamus, the reticular formations of the neuraxis and thence through the autonomic nerves, it imposes upon the reflex and humoral automatic activities of the viscera a general directive pattern of control. This, together with the associated pattern of voluntary muscular behaviour, constitutes the coordinated response of the individual to the demands of his internal and external environments.

Functional or psychosomatic disorder occurs in any individual when the cortex fails to educe an harmonious pattern of visceral behaviour perfectly adapted to both internal and external environmental demands. This may happen when there is insoluble conflict between the demands of the inner man and the external environment, or between two irreconcilable demands from without. It may also occur when the cortex, through toxæmia, neuronal poverty, or other cause, is incapable of dealing with problems easily soluble under normal conditions. In any case the physiological hallmark of such disorders is their origin in autonomic nervous interference, of cortical origin, with the efficient function of any organ or group of organs.

The normal cortical interferences with, or adjustments of, visceral behaviour are manifold and complex, but certain patterns of commoner type will serve for illustration. The effort reaction is a nervous preparation of the body for effort involving autonomic stimulation of cardiac and respiratory action, glycogenolysis, deviation of blood from the viscera (stomach, bowel, renal cortex) to the coronary-skeletal-pulmonary circuits, nervous sweating and the like. When this is roused as part of a fear or flight reaction, digestion is inhibited; when as part of an aggressive or fight reaction there is on the contrary face-flushing, salivation and gastric hypermotility, hyperæmia and hypersecretion, even though the stomach be empty.

The well-known psychic preparation of the stomach for digestion in response to food signals from without, and the intensification or suppression of these preparations by hunger, repletion or nausea within, need no comment.

A parallel mass preparation is made for sexual activity. The cortical influence over bladder and rectal activity and sleep, and the widespread autonomic changes occurring in laughter or weeping, need only be mentioned.

Functional or psychsomatic disorders arise in several ways. First there are the normal minor disturbances inseparable from civilized existence, except in the rare completely balanced human being. The ordinary run of human cortex does not, except after considerable experience, answer an environmental call for sudden adjustment of visceral function in an accurate, specific and precise manner. Anxiety or resentment over matters which are not to be solved by immediate physical exertion nevertheless produce the cardiac, gastric and bowel adjustments proper only to fight or flight. The normal cortex certainly, when faced with situations which it has previously encountered, will adjust better and better with practice. Happy, however, is the man who does not at some time suffer nausea, diarrhœa, palpitation, insomnia, migraine, dyspepsia, asthma or the like, in the face of a strange and worrying situation.

The more severe degrees of functional disturbance occur when such environmental disturbances are beyond the power of the individual to solve, either, as previously noted, because of the nature of the situation or because of a poorly endowed or toxic neuronal equipment. Here it is clear there may be prolonged visceral disturbances, perhaps of anxiety type with effort and hyperventilation syndromes, perhaps of chronic dyspepsia, of impotence or frigidity, of insomnia, constipation and the rest.

Under such circumstances two sets of further changes may occur. The first set occurs in the cortex where, as Pavlov showed in his dogs, over-stimulation, over-prolonged calls for strong inhibition, or acute conflicts, may give rise to breakdown in which the cortical capacity for graded response to quite ordinary stimuli is lost, and the organ thereafter responds non-quantitatively, paradoxically or hyperparadoxically instead of in its former physiological manner. The likeness of these experimental neuroses in animals to human anxiety states and other neuroses cannot be ignored clinically.

The next set of changes occur in the over-stimulated or over-inhibited viscera themselves and thence secondarily in the whole metabolic state of the individual. An organ which has chronic alteration in its activity or blood supply imposed upon it as part of a general behaviour reaction, to the detriment of its normal local function, tends to become physiologically demoralized.

Changes occur in its resistance to infection and trauma, in the threshold of its sensory and organ innervation, and thence in the synaptic relations of its afferent representation in the central nervous system. Anæsthesias, paræsthesias, or hyperasthæsias develop; inflammatory or traumatic injury may be facilitated. Finally such dis-

[1] Part of a symposium on "The Psychosomatic Approach in Medicine", read at a meeting of the Queensland Branch of the British Medical Association on July 2, 1948.

turbances produce general repercussions. In the effort syndrome, the stomach, for instance, is kept in a state of constant inhibition while the respiratory apparatus is continually stimulated to tachypnœa, both in a manner which has no reference to any possible, even temporary, expression in physical flight. It is clear that subclinical tetany and chronic nausea or dyspeptic pain (even, in the later stages, ulceration of the gastric wall) may be the natural and inevitable outcome, with all the secondary disturbances that these automatically bring in their train.

For these types of disorders the term functional seems the least objectionable; but, it is clear, such functional disturbance usually involves some weakness or abnormality of the cerebral neuronal capacity in the first instance. Secondly, there is little doubt that organic changes—for instance, hæmorrhoids, pelvic infections, coronary spasm and degeneration, gastric and duodenal ulceration, gall-bladder stasis and even hypertensive circulatory disturbances of the kidney—may evolve from origins purely functional. Such functional derangements, moreover, if occurring as complications in organic disease, may do even more to increase suffering and shorten life than the actual physical disease process itself.

The first duty of a physician to his patient is to be able to recognize the causes of things. Thus equipped he may perhaps cast out fear, defeat inexorable fate and halt the greedy, roaring flood of pain. Happy he, as the poet remarked, who is able so to do. This, however, is impossible unless he understands the mechanisms by which normal function and structure may be perverted and diseased by untoward physiological and neurological stresses without any primary intervention of any organic disease process.

The problems involved, as Cannon perceived and stressed, are problems of clinical physiology. Unless the approach is physiological there is little hope of being able to assign correctly the symptoms and signs of any individual patient to their primary functional or organic cause. Unless this is recognized the same old sad mistakes will continue; the mistake of regarding symptoms as imaginary, even assumed, because no organic cause for them can be found; the mistake of regarding the discovery and treatment of some or any organic disease process as the be-all and end-all of the physicians' art; and finally the common, grievous and cruel mistake of afflicting a sufferer from a functional disorder with a fear-charged diagnosis of non-existent or irrelevant organic disease.

PSYCHOSOMATIC ASPECTS OF SURGERY.[1]

By ALAN E. LEE, M.D., F.R.C.S. (England),
Brisbane.

THOUGH at the present time the term psychosomatic is often limited strictly to those situations in which continued emotional tension has resulted in the production of structural body changes, I propose to apply the term in a much wider sense.

Prior to the production of these structural changes, there has been a long intermediate stage in which the emotions have been effecting functional changes which eventually result in the physical defect.

I would ask permission then to regard as psychosomatic all the bodily signs and symptoms that are produced when emotional tension, instead of finding its normal outlet in rage or tears, fight or flight, becomes repressed and short-circuited into somatic channels, where the emotional energy is expended in symptom production.

It may be at once objected that the province of the emotions is not the concern of the specialist surgeon but of the psychiatrist. I would say, however, in answer that the unity of the body and mind is such, and the influence of emotion so all-pervading, that in no department of medicine, however specialized, can it be neglected either in diagnosis or in treatment.

It becomes increasingly evident that no specialist, unless he is in addition a good physician with some psychological knowledge, can safely manage patients coming within the limits of his specialty. Every patient must be regarded as a whole person, and both his body and his personality be examined, before the cause of symptoms can be stated accurately.

If it be further objected that the medical profession is at present quite untrained to exercise such a function, one can only reply that a most urgent indication exists that this defect in its knowledge should be corrected. Basically, much of the pressure for a revolutionary change in the organization of medical practice is based on an awareness of this fatal chink in our professional armour.

Though the early steps in the closing of this defect must be the proper training of an immensely larger number of psychiatrists than exist today, and their diffusion through all departments of medicine, the ultimate goal must be the training of all medical students, at every stage of their clinical studies, in the appreciation of the all-pervading influence of emotion, and in the nature of the syndromes that are so produced. The general surgeon and the general physician, equally with the psychiatrist, must play their part, by example and by teaching, in this new approach to the patient.

I would now like very briefly to state those ways in which the short-circuiting of emotional tension into bodily channels impinges upon the practice of the abdominal surgeon.

Till very recent times, and still in most surgical text-books, the syndrome of a disease has been related entirely to its ætiological stimulus, whether this is traumatic, infective, or what not, and the reaction of the patient has been assumed to be constant and unvarying from person to person. But this newer approach in medicine shows that the bodily reaction is by no means uniform and that intense variations in the receptor mechanisms occur; these variations, moreover, are largely activated by the short-circuiting of emotional tension.

Equally important in this relation is the recognition of a threshold in symptom production, so that a certain level of intensity must exist before a stimulus produces a reaction that rises to perception. Apart altogether from stimuli from without, an immense number of activities are constantly occurring within our bodies, mainly physiological in hollow organs, and are controlled by the autonomic nervous system, that in a person of average threshold never rise to consciousness.

The commonest cause for a lowering in the sensory threshold is emotional tension, and the production of psychosomatic symptoms of the functional type—that is, those unassociated with structural change—consists largely in the bringing to perception of these usually subthreshold physiological activities.

The discomforts produced may be classed as examples of organ awareness. Though this is a readily understandable phenomenon, it is curious how often doctors fail to appreciate the nature of the symptoms so produced.

That palpitation is an awareness of the beating of the heart against the chest wall should be pretty obvious. Nor when immediately after a meal there is a complaint of epigastric fullness should there be any difficulty in appreciating that the patient is simply aware of the tension of his full stomach. But because the patient rather naturally attempts to interpret the discomfort as due to wind, or flatulence, or fermentation, or to certain foods disagreeing with him, many doctors are tempted to embark upon a will-o'-the-wisp search by much ancillary investigation to find a physical abnormality where none exists, and thereby leave the patient not only poorer but more uncertain and anxious than before as to the cause of a perfectly simple symptom.

You need only turn to the pages of an excellent book like Alvarez's "Nervousness, Indigestion, and Pain", a book which every practising doctor should read and reread, to find listed all those physiological functions in hollow organs which, granted a sufficiently low threshold, can rise to perception as a discomfort.

[1] Part of a symposium on "The Psychosomatic Approach in Medicine", read at a meeting of the Queensland Branch of the British Medical Association on July 2, 1948.

To elucidate any problem of organ awareness, you must first know the physiological function of the organ, secondly understand clearly the mechanism by which such an organ can produce symptoms, and thirdly learn how to assess the level of an individual's sensory threshold.

I would stress particularly that such a diagnosis can then be a positive one—not made only after eliminating all possible physical explanations, but by recognizing that this is a psychosomatic syndrome, and can be nothing else.

So far I have discussed organ awareness as the perception of normal function in normal organs. But emotional stress also acts frequently in the sense of altering normal physiological function, largely via the mediation of the autonomic nervous system.

In this way, as applied to abdominal organs, variations in smooth muscle tone and in secretory activities may be expected. Though such emotional dysfunctions are common, I am doubtful if they are often responsible for pain production. It is, however, such a disordered function that may express itself later in structural changes.

Thus the vagal-mediated rapidly emptying and freely secreting stomach, in which the secretion is occurring apart from the stimulation of food-intake, is in the modern fashion, and with at least some truth, regarded as the precursor of chronic peptic ulceration.

The apprehension associated with an X-ray examination produces the pylorospasm, the gastric stasis, and the duodenal irritability which can so easily result in a misdiagnosis of duodenal ulcer.

The simultaneous occurrence in the gall-bladder of an episode in which increased mucoid secretion is associated with a rise in the bile salt content produces those functional changes which can result in the production of a family of primary gall-stones, and at least one distinguished observer believes that the common stimulus to such a functional change is emotional tension.

In the colon, the excessive secretion of mucus and the muscular irritability produce chronic diarrhœa. If such a functional state becomes associated with the mucosal œdema of allergy, or with the effects of infective inflammation, ulcerative colitis may occur.

Though these motor and secretory changes that are produced by autonomic nervous imbalance as the result of emotional tension are important, I am even more interested as an abdominal surgeon in the relation to differential diagnosis of the frequent superficial neuralgias that are so common an expression also of the same nervous tension.

In the few remaining minutes at my command I cannot begin to discuss the complex mechanisms that underlie such neuralgia production and must be content with some dogmatic statements.

Firstly, though these pains rise to perception as the result of lowering of the sensory threshold by emotional tension, yet they are all the expressions of a local stimulus. Usually this is not purely physiological but mildly abnormal, as, for example, a visceral drag in the erect posture in a person with a non-fixed right colon. But before the stimulus can rise to perception it must have recurred frequently over a long period of time, so that a synaptic facilitation has occurred, while in addition there must exist not only a general lowering of the sensory threshold, but often also as the result of a local anxiety fixation this particular facet of the body image in the sensory cortex must be delineated with especial vividness.

Secondly, abdominal wall neuralgias can be separated from other varieties of abdominal pains in that: (*a*) they have the character of surface pain, sharp, stabbing, burning or aching, and accurately localized; (*b*) they are distributed accurately along dermatomes; (*c*) they are associated with tenderness in their distribution, with a maximum soreness where the sensitive nerve pierces the outer sheath of the rectus muscle; and (*d*) they are usually associated with muscular hyperæsthesia in their distribution. Abdominal neuralgias are essentially lateral pains, and it may be stated broadly that the presumption in the case of all chronic lateral pain is that it has an emotional origin. Even when acute attacks occur this is still substantially true, even though such important physical entities as the neuralgic component of renal colic and the abdominal wall extensions of the inflammations of acute appendicitis and acute cholecystitis have to be excluded. As a very important corollary the deep pain both of gastro-intestinal origin and of pelvic disease is essentially mid-line. An enormous amount of unnecessary surgery has rested on non-recognition of this fact. It is not complimentary to the standards of the Australian medical profession to have to state that of patients presenting themselves with abdominal pain on whom a previous abdominal operation has been performed, in not more than one case in five was the former operation based on sound grounds, nor did it succeed in removing the symptoms for which it was performed. And nearly always this later survey will show that the symptoms unrelieved by operation were essentially lateral neuralgic pains of emotional origin.

To sum up this necessarily sketchy statement of psychosomatic disturbances as they affect the abdominal surgeon, I would point out that such conditions are extremely common; as someone has said, the abdomen is the sounding board of the emotions. Repressed emotional tension reveals itself here in three main ways: by producing organ awareness; by causing increased autonomic activity that expresses itself first in motor and secretory changes, and later in structural disease; and in the production of abdominal wall neuralgias.

Such conditions can be positively recognized, and not assumed only after a process of negative elimination, and their management is well within the competence of the soundly trained physician with some psychological insight. And since these conditions represent the majority of all abdominal complaints, it can never be otherwise, unless some radical change occurs in the whole medical set-up, whereby psychiatrists will represent the main body of the practising profession, while the treatment of physical ills becomes relegated to a minority group of specialists to whom can be referred cases outside the scope of the psychiatrist's competence.

PSYCHOSOMATIC ASPECTS OF PÆDIATRICS.[1]

By DAVID JACKSON,
Brisbane.

IN contributing the point of view of a pædiatrician to this symposium, I want, at the outset, to make it clear that I am speaking as a clinical pædiatrician and not as a child psychiatrist.

The very nature of a pædiatrician's work demands that, if he uses the word "psychosomatic" at all, he must use it in a much wider sense than has been done in either of the previous papers, namely in the sense that we are treating a human being, a patient rather than a disease—the patient as a whole, and not only the physical effects of his illness.

Rather than talking of psychosomatic disorders, as if some can be so classified and some not, we know that the correct use of the word is in the title of our discussion: "The Psychosomatic Approach in Medicine". It is the approach that is important—the bearing in mind that both psyche and soma are affected, and the deciding to what extent each is involved in a particular problem. This is just as important in treating children as in treating adults, though the approach may be rather different, for we are dealing with a young animal in the developing stage, with a body whose development we can favour or prejudice, but also with a developing mind that is in a plastic state, influenced for good or bad by all sorts of factors in the child's environment, and very much at the mercy of the intelligence of those into whose care it is given.

Strangely enough this is very generally recognized, though less often remembered, in medical practice. Thus

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we all associate many, perhaps too many, complaints with Monday morning school, and all of us must have said of many children that the trouble is less with the child than with the parents; in fact it has almost become a platitude to say that there are no problem children but only problem parents.

On the other hand it may be said, and in fact very often is said, that what makes the treatment of children such a pleasant pursuit is that we are treating what might be called "pure" diseases, and that the clinical picture, progress and recovery are uncomplicated by that dreadful miasma of emotional factors which so often overlies physical disease in adults. And certainly it is true that many of the classical illnesses of childhood as described in text-books and seen in children's hospitals are sudden calamities, generally due to infection, that strike a healthy, happy, and apparently normal child (such as pneumonia, pyelitis, nephritis, poliomyelitis, osteomyelitis, and even rheumatism). In infancy the preoccupation with the disease and its treatment is perhaps even greater, for, while work on fluid and electrolytic balance has wrought great changes in treatment, and while we deal with charts, formulæ and biochemical estimations and, "observing some chemical excess, will deftly neutralise it with a pinch of salt", it cannot be denied that there is a tendency for the patient to become as impersonal to us as is a system of tubes and bottles on a laboratory bench.

But even in these apparently straightforward cases, in which emotional or psychological factors seem to be absent from the ætiology, we cannot ignore the possible effect of the illness, or of our management of it, on the child's future mental development, that is, on the personality with which he will have to face life in the future. And quite apart from these cases there is a large group in which the relationship of emotional, personality, and environmental factors to the physical manifestations or presenting complaint is extremely complex. One meets such cases frequently in specialist practice, the patient having been either referred by a doctor after a painstaking investigation has failed to find any cause for the symptoms, or brought by parents who have tried doctor after doctor and treatment after treatment.

Now we cannot, in a discussion of this nature, attempt a catalogue of children's diseases, giving the possible psychological accompaniments and sequelæ of each, nor can we discuss at any length the psychopathology of childhood. But in order to get a rapid general view of the type of problem that we may meet, a classification such as that suggested by Kanner is helpful. There are three groups: (*a*) that in which personality difficulties express themselves clearly as whole-dysfunctions of the individual; (*b*) that in which personality difficulties express themselves in the form of involuntary part-dysfunctions; (*c*) that in which personality difficulties form essential features or sequelæ of physical illness.

The first group is a large one, but in most instances the psychological element is so obvious that its recognition will cause little difficulty, as in the mental defects, emotional disorders (such as temper tantrums), anti-social trends (disobedience, lying, stealing), disorders of sleep, and even the relatively rare psychoses. But it also includes one of the commonest difficulties about which we are consulted, namely, faulty feeding habits and, in particular, lack of appetite. In many, perhaps even the majority of cases, this anorexia is an expression of general contrariness in a spoiled and overprotected child, and is the result of, and is maintained by, parental mismanagement. Very often we can trace the beginning of the trouble to bad management in infancy, such as too long on breast or bottle and a difficult weaning, and in addition I must stress what may be called the "weight-consciousness" of many mothers which leads them to force food into a child literally *ad nauseam*. This may begin in infancy, often when a temporary illness makes a child disinclined for food, but this fear of starvation, or fear that her child will not grow, persists with a mother for a very long time.

The second group gives more difficulty, and it is often very great difficulty, for here we have localized disturbances of function, which seem to be limited to a specific organ or system. A common feature is the very significant fact that, in spite of adequate investigation, organic lesions cannot be demonstrated. While it must be remembered that such an investigation is absolutely necessary, many of these problems can best be understood and treated by studying the patient in his entirety, with attention to physical, emotional, constitutional, and environmental peculiarities.

I may mention, as an example, headache. If this is not an accompaniment of physical illness, it may be due to fatigue, especially that from a constant strain such as occurs in a child expected to do school work beyond his capacity. In such a case it is not enough to enquire, of parent or child, whether he is good at school, or enjoys school. A much more careful investigation is necessary, for often it is the bright child, striving to maintain his supremacy or a place really beyond his capability, who is affected. A different type is the "headache of convenience", and here the distinction from frank malingering may be difficult. However, it is interesting to find how often this refuge from some unwelcome activity is acquired from a mother who has the same habit. Psychogenic vomiting is a common response to personality difficulties, but one must be very sure that there is no underlying organic cause, and the same applies to constipation and diarrhœa. An obsession on the mother's part about regularity or cleanliness is easily passed on to the child, perhaps in a distorted form, or faulty training may cause trouble later on, but again we must be careful in our investigation. The same is true of enuresis, which also belongs to this group. Most cases are due to faulty training or to some non-organic cause, even if it be simply very sound sleep, but proper investigation is essential. It is interesting to note that complaints related to the heart, such as pain around the heart or palpitations, are relatively rare in childhood, perhaps because few children attach the same significance to this organ and region as do adults. If such complaints do occur they may be due to parental influence or suggestion. Respiratory symptoms seem to be rare as expressions of psychogenic part-dysfunctions, though a cough may persist when it has brought a child special attention, and become almost a habit.

In the third group there are the personality changes following organic cerebral disease such as encephalitis or juvenile general paralysis of the insane, and those associated with infections or toxic illness. There are the personality features of endocrine dysfunctions, the excitability of hyperthyreoidism and the placidity and mental retardation of hypothyreoidism, for example. Here we have a "reversal", that is to say, an abnormal behaviour with a somatic basis.

In this group, also, we must include the effects or possible effects of prolonged illness or hospital treatment, as is well shown in cases of rheumatism, and also in the curious and distinctive personality which develops in association with cœliac disease. I am sure we must remember this in our whole approach to and management of a child with a prolonged illness. We must remember it when, perhaps, we keep a child in hospital for an unnecessarily long time, when we are discussing his illness in his hearing, or when we are examining him day after day or arranging a series of investigations, for we never know when we may be fixing an imaginative child's ideas on a particular part or organ, and perhaps laying the foundation of "flight into disease" later on. We must remember it even when dealing with infants in the first year—the effect of separation from the mother, and the possible effect of the illness on the mother's subsequent attitude towards the child. And I cannot leave this aspect of the subject without a plea that it be remembered when orthopædic splints and appliances are being considered.

In conclusion I want to make three observations on the treatment in these cases. The first is to emphasize its difficulty—in the sense that to identify a problem is not to solve it, and that a great deal of trouble and patience, and often tact, is necessary both to discover the true facts of each case and to set them right. Personally, I find no greater discouragement in any aspect of pædiatric practice than in this task of trying to find out what the home

conditions and family background are really like, and then in setting about persuading the parents to make what may be a most sweeping change in their whole mode of living and in the running of their home. Even with intelligent parents who seem to appreciate and understand the position as we explain it to them, we are only too often left wondering just what does happen in the home when, so to speak, the door is shut. But these difficulties and discouragements, great though they may be, are no reason for not trying to do what we can.

Secondly, I would stress the importance of correcting any physical disorder, even if it is not an obvious component of the presenting complaint. Even if we decide that a child's trouble is due primarily to emotional or environmental factors, we will have lost our balanced approach if, in correcting these, we neglect to treat, say, incidental anæmia or carious teeth.

Lastly, the scope of preventive (or directive) treatment, here as elsewhere in the pædiatric field, must be emphasized. We see children because they are ill (or the parents think they are), and all too rarely do we see children periodically when they are well. But at all times we must remember the unseen factors of home and family, school, work and play, and in order to have a true picture of our patients as a whole, we should know and understand as much about them as we can. Then perhaps we can detect trends which may be corrected and, if we can but realize that many manifestations of disease in childhood are either actually associated with faulty adjustment or may lead to it, surely we can do much to prevent serious disorders and even catastrophes in adult life.

THE DIAGNOSIS OF EIGHTH NERVE TUMOUR.[1]

By GILBERT PHILLIPS,
Sydney.

THIRTY years ago Harvey Cushing[1] described his Baltimore and Boston series of tumours of the *nervus acousticus* in a monograph which still remains an authoritative text on this subject. The series of thirty cases then described represented 6% of the total 468 verified intracranial tumours, the series being sufficiently large for Cushing to make the point for the first time that nearly 50% of posterior fossa tumours were extracerebellar, and that of these approximately two-thirds were tumours of the eighth nerve. Until this time the operative mortality rate had been very high. Borchardt[2] in 1906 reported a mortality rate of 72·2% and von Eiselsberg[3] at the International Congress in 1913 one of 75%; at the same meeting Krause,[4] whose name has become associated with the unilateral suboccipital operation, declared that his personal operative mortality was 33·3%. A great drop in these figures was seen in Cushing's surgical cases, the mortality in the earlier Baltimore series being 36·3% and in the later Boston series 10·5%. This represented the mortality for the intracapsular technique at that time, and more recent developments in surgical technique have now established a considerable reduction in this last mortality figure. Perhaps the most interesting advance which followed Cushing's monograph was the new approach to diagnosis which resulted from his emphasis on the definitive chronological order in the appearance of symptoms with early eighth nerve involvement and later compression, in a now well-recognized sequence, of the adjacent cranial nerves in the cerebello-pontine angle.

In this article it is intended, however, to emphasize an important point which was made by Cushing, namely, that in spite of a recognized chronological order of symptoms during the growth progress of an eighth nerve tumour, many cases are atypical and the predominance of a single symptom or sign out of its usual sequence not infrequently leads to misinterpretation and incorrect diagnosis. The list of erroneous diagnoses may include Ménière's syndrome, Bell's palsy, clonic facial spasm, focal epilepsy, trigeminal neuralgia, occipital neuralgia, torticollis, syphilitic meningitis and subdural hæmatoma, all of which may be relatively common errors and could be added to by a much greater list of rarer lesions. If we realize that approximately one in every fifteen intracranial tumours is a tumour of the acoustic nerve, it would seem important that some guide should be given to the clinician in the early establishment of a diagnosis in these atypical cases, so that surgical treatment may be instituted before a high level of intracranial pressure is established. It is hardly necessary to emphasize the fact that the surgery of the cerebello-pontine angle provides a most difficult problem for the operator. Since he must work in a small field of considerable depth, hemmed in on all sides by large and numerous arterial and venous communications, in an attempt to deal with a lesion which itself may be extremely vascular and which in most cases has already caused compression of adjacent medullary centres, the merit of establishing the final diagnosis before the intracranial pressure is raised must immediately commend itself to the operator.

My personal experiences of this neoplasm have confirmed Cushing's early statement, but it is felt that a particular grouping of signs can usually be identified at an early stage in these cases, and that this simple pattern in the clinical picture may permit a confident diagnosis of an eighth nerve tumour. It is intended now to illustrate with a series of my own cases some of the difficulties and errors in early diagnosis, and to emphasize the fact that the patient with an eighth nerve tumour may not be deaf in that ear and that the single symptom or sign which predominates may distract the observer's attention from the cerebello-pontine angle.

CASE I.—E.M., a female, aged fifty years, was referred with a diagnosis of Bell's palsy. She had previously been an active healthy woman and had never suffered any serious illness. There was no history of head injury and there had been no previous operations performed. She awoke one morning, and on looking at herself in the glass found that the right side of the face was completely paralysed. She complained of a feeling of stiffness and numbness on the right side of the face, but had no other symptoms. Neurological examination revealed normal fundi. There was no intracranial bruit. She had a partial nerve deafness on the right side, which she stated had been present for many years. There was no history of tinnitus, giddiness or ataxia. The corneal reflex on the right side was diminished compared to the left. These findings led to the suspicion of a cerebello-pontine angle lesion and further investigation revealed the absence of a caloric reflex on the right side, and cerebro-spinal fluid with a pressure of 200 millimetres of fluid and a protein content of 120 milligrammes *per centum*. The final diagnosis of right acoustic nerve tumour was made, which was subsequently confirmed at operation.

CASE II.—L.G., a male, aged thirty-nine years, was referred as a probable subject of Ménière's syndrome. He stated that for approximately two years he had had progressive deafness in the left ear which had been accompanied by tinnitus and severe attacks of giddiness. He had previously been treated for some time as a subject of Ménière's syndrome, and an exploratory mastoid operation also had been performed. Further careful inquiry elicited the fact that he had experienced numbness and tingling in the left side of the tongue during the past six months. Full neurological examination did not reveal any abnormality apart from a partial nerve deafness on the left side. It was found that the left caloric reaction was absent and the cerebro-spinal fluid which was under a normal pressure of 120 milligrammes of fluid contained 120 milligrammes *per centum* of protein. A left suboccipital craniotomy was performed and an acoustic nerve tumour removed after left hemicerebellectomy.

CASE III.—E.I., a female, aged forty-eight years, had complained that for four years she had been unsteady on her feet and was inclined to deviate to the right on walking. Her ataxia was increased under conditions of poor illumination, and an early provisional diagnosis of *tabes dorsalis* had been made. Later it was suggested that the condition was due to peripheral neuritis, and she had been treated for this condition in hospital. Further inquiry revealed that during the past two years she had noticed deafness in the right ear and subjective numbness at the right angle of the mouth and on the right side of the tongue. It was found that she had no caloric reaction on the right side, and her

[1] Read at a meeting of the honorary medical staff of the Royal Prince Alfred Hospital on August 10, 1948.

cerebro-spinal fluid, which was under a normal pressure of 110 millimetres of fluid, contained 150 milligrammes *per centum* of protein. At operation a right acoustic tumour was disclosed and removed.

CASE IV.—S.L., a male, aged forty years, had been referred to a physician complaining of generalized headache for six months. His condition was monosymptomatic, and he was informed that it was "nerves" and was given a tonic. When seen by me he stated that during the past six months he had also had some slight deafness in the left ear associated with tinnitus. Further examination also revealed a diminished left corneal reflex, hypæsthesia of the left side of the face, ataxia in the left hand and a relative nerve deafness on the left side. Caloric reaction was absent on the left side and the cerebro-spinal fluid, which was under a normal pressure of 140 millimetres of fluid, had a protein content of 400 milligrammes *per centum*. A left acoustic tumour was exposed at operation and removed.

CASE V.—A.M., a female, aged forty-eight years, had complained of severe pain on the left side of the head for six months and, as there was a history that she had fallen down a staircase a few weeks before the onset of her pain, she was referred to me as a probable subject of subdural hæmatoma. Further inquiry revealed the fact that her pain in the left temporal region also extended forwards to involve the left side of the nose and the left angle of the mouth. She also stated that she had noticed some slight unsteadiness on walking during the past twelve months. This patient had been aware of deafness in both ears for some years and had been contemplating a fenestration operation. Further examination revealed the absence of a caloric reaction on the left side, and her cerebro-spinal fluid, which was under a pressure of 220 millimetres of fluid, had a protein content of 150 milligrammes *per centum*. At operation, following left hemicerebellectomy a large eighth nerve tumour was removed.

CASE VI.—R.B.B., a female, aged fifty years, was aware that she had been deaf in the right ear for years, but had been perfectly well and able to carry on her occupation as a draftsman until twelve months previously, when she commenced to experience severe pain in the back of the neck. The condition had been diagnosed as fibrositis, and she had been treated for some months by injections. During a refraction estimation the ophthalmologist thought that the ocular fundi showed early papilloedema, and further inquiry revealed the fact that the patient had noticed a feeling of thickness and stiffness at the right corner of the mouth for one month. In view of the trigeminal involvement, a cerebello-pontine angle lesion was suspected and it was found that she had no caloric reaction in the right ear. The cerebro-spinal fluid was under a pressure of 260 millimetres of fluid and contained 150 milligrammes *per centum* of protein. At operation an acoustic nerve tumour was exposed and removed.

CASE VII.—R.P., a male, aged fifty-nine years, had been deaf in the right ear for twenty years. In 1939 he had a severe attack of vertigo associated with vomiting and a second attack occurred in 1943. He was seen by an otologist, who treated him for some time as a subject of Ménière's disease. Further inquiry revealed that he had not suffered from tinnitus or headache. However, his right corneal reflex was diminished and he was found to have no caloric reaction in the right ear. The cerebro-spinal fluid was under a pressure of 250 millimetres of fluid and the protein content was 200 milligrammes *per centum*. At operation on April 3, 1946, no tumour was seen in the cerebello-pontine angle, but a large cystic collection was found enclosed in what appeared to be an adhesive arachnitis. This was removed. The patient's condition improved after operation, but a later check of his cerebro-spinal fluid revealed the fact that the pressure remained high and that the protein content was progressively increasing. It was decided to operate, and on April 2, 1947, the right cerebellar hemisphere was removed and a medially placed acoustic nerve tumour exposed and removed.

CASE VIII.—N.A., a female, aged fifty-five years, complained of weakness of the left arm and left leg for three years. During the past eighteen months she had also had a series of "blackouts" which resembled syncopal attacks. The condition was regarded as being due to some vascular pathological state, and twelve months previously she had had an operation for termination of pregnancy for this reason. When examined by me she was found to have hypæsthesia of the left side of the face and a diminished left corneal reflex. There was relative nerve deafness of the left ear and no left caloric reaction. She had early bilateral papilloedema. The cerebro-spinal fluid pressure was 340 millimetres of fluid, and the protein content 90 milligrammes *per centum*. At operation, after partial cerebellectomy a left acoustic tumour was exposed and removed.

CASE IX.—A.C., a female, aged thirty-one years, had noticed that she had had a regular increase in weight over the past eight years and had gained three to four stone in that period. Some five years ago she noticed that she had slight deafness in the right ear and subsequently experienced episodes of tinnitus on that side. There had been no vertigo and no diplopia. She had become depressed and anxious recently. On examination, she was found to have bilateral papilloedema and slight deafness in the right ear. There were no other abnormal neurological signs. X-ray examination of the skull showed an erosion of the internal auditory meatus on the right side. The cerebro-spinal fluid was under a pressure of 300 millimetres of fluid and the protein content was 250 milligrammes *per centum*. Caloric reaction on the right side was absent. At operation a right-sided eighth nerve tumour was exposed and removed.

The foregoing series reveals that these patients may not be deaf or may not be aware of unilateral deafness if it is not associated with tinnitus, or alternatively, that the unilateral deafness may have been present for so many years before the appearance of other signs that it is not regarded as significant in the later clinical picture. The wide range of diverse leading symptoms and signs is also shown, and these may include giddy turns, weakness of the extremities, headaches, pain in the neck, facial paralysis or ataxia. It is believed that these atypical cases, however, have a common group of signs and that, if this common pattern of disturbance is sought for and identified, the diagnosis of acoustic nerve tumour may be made. If the caloric reaction is absent or markedly depreciated, if there is a considerable rise in cerebro-spinal fluid protein content and if there is evidence of subjective or objective disturbance in the trigeminal field, the lesion is almost certainly a tumour of the eighth nerve. It has previously been pointed out by several observers that the vestibular nerve and not the cochlear portion is the site of origin for the eighth nerve tumour, and it may be expected, therefore, that the caloric reaction will be interfered with at a very early stage in the growth of the mass. Cushing[4] has remarked that the cranial nerve most frequently involved after the eighth is the trigeminal, and in many cases specific inquiry must be directed to this point, as the patient will often dismiss a feeling of numbness or tingling at the angle of the mouth as having no relation to the main complaint. Moreover, there may be no subjective trigeminal involvement, and it is always necessary therefore carefully to compare the corneal reflexes on the two sides. The third point lies in relation to the cerebro-spinal fluid protein content and acoustic nerve tumours. Phillips and Goswell[5] have reported in a series of 161 intracranial tumours that there was an average increase in the protein content to 120 milligrammes *per centum* in every acoustic tumour which was verified at operation. This relationship is firmly established by other observers, and an increased protein content is constantly associated with acoustic tumour. It is suggested therefore that the triad of trigeminal defect, absent caloric reaction and increased cerebro-spinal fluid protein content establishes the diagnosis of a tumour of the eighth nerve.

An interesting point to note in relation to the series of cases reported here is that all the patients are alive and well, some many years after operation, and this fact may be correlated with relatively early diagnosis and consequently operation under conditions of only slightly increased pressure. In only one case in this series was there any considerable increase in intracranial pressure. Herein lies the importance of recognition of the pattern of definitive disturbances described when the patient is first seen.

Summary.

1. A patient with an eighth nerve tumour may not have a unilateral nerve deafness or, if it is present, he may not be aware of it.

2. A leading symptom, not specifically related to the cerebello-pontine angle, may be misinterpreted and lead to incorrect diagnosis and treatment.

3. The delay resulting will increase the hazard to the patient and the surgical difficulty, owing to the development of increased intracranial pressure.

4. The combination of trigeminal nerve involvement, absent caloric reaction and increased cerebro-spinal fluid protein content forms a secure basis for the diagnosis of a tumour of the eighth nerve.

References.

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(3) A. von Eiselsberg: "Über die Chirurgische Behandlung der Hirntumoren", "Transactions of the International Congress of Medicine, London, 1913", Section VII, page 203.
(4) F. Krause: Discussion of von Eiselsberg's paper,(3) *loco citato*.
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SOME ASPECTS OF SUBACUTE BACTERIAL ENDOCARDITIS.[1]

By E. F. GARTRELL,
Adelaide.

ALLOW me to assure you that I appreciate the privilege of addressing you this evening; but lest I should be taken to task over my choice of subject, I had better make an explanation. I admit the rarity of subacute bacterial endocarditis, which is found in only 1% to 2% of all cardiac patients; but, on the other hand, it is a complication in 8% of all congenital heart conditions and in 5% of rheumatic conditions. This, however, is not the most cogent reason for our interest in the disease, for the therapeutic triumph of penicillin in transforming a virtually incurable complaint into one amenable to treatment has set up a milestone on the *via therapeutica*. By so doing it has placed on our shoulders the responsibility of curing the majority of these patients of their infection. In saying "curing them of their infection", I speak advisedly, for, although we can do this in most cases, quite an appreciable percentage die from heart failure, uræmia, emboli *et cetera*, so I would also direct your attention to the task of reducing this wastage.

Kirkes in 1852 gave us the first clinical picture of this condition. In 1873 Rapin noted those lesions which we now know as Osler's nodes; but it was not until 1885 that Osler himself pointed out their real significance in the course of his comprehensive review of the disease. Of course, one cannot deal with the subject as a whole in one short session; but there are two main aspects which I wish to present to you—namely, (i) diagnosis, and (ii) treatment. The results of treatment are intimately dependent upon the speed with which diagnosis is accomplished; but I propose to pay more attention to treatment, in an endeavour to assist the general practitioner in dealing with the average case.

Special cases demand special measures which can be adopted only in a hospital with adequate laboratory facilities, and by the closest cooperation between physician and pathologist. I thank the honorary physicians of the Royal Adelaide Hospital for their courtesy in allowing me to review their records. The numbers, of course, are too small to be of statistical significance; but there have been many incidents of interest about which we hope to hear in the discussion.

Diagnosis.

With regard to diagnosis, I shall not seek by a surfeit of symptoms to soothe you into a state of somnolence, but I propose to give you a few main branches on which to develop a diagnostic tree. The four cardinal features in the fully fledged condition are: (i) evidence of a valvular or congenital cardiac lesion, (ii) pyrexia, (iii) embolic phenomena, and (iv) the culture of microorganisms from the blood. In addition we note that this is a disease mainly of young people; but let us not forget that in some series 4% of patients have been over the age of sixty years. A history of dental sepsis or extraction may be obtained in a number of cases. In amplification one can say that rheumatic valvular disease of the heart, and often the less severe involvement rather than the gross stenotic lesions, forms the starting point in the majority, changes in the murmurs being suggestive events. The pyrexia varies from patient to patient, and is evidence of the toxæmia which produces other symptoms and signs including malaise, weakness, aches and pains in limbs or joints, anorexia and loss of weight. Blood changes include secondary anæmia, an increased blood sedimentation rate and a raised leucocyte content. This rise is often not above 10,000 per cubic millimetre, but the percentage of polymorphonuclear cells is usually raised. A "false positive" Wassermann reaction is sometimes obtained. One sign sometimes found is tenderness of the sternum to percussion. The embolic phenomena are frequently the outstanding features, and so may be the pointers which lead us to the diagnosis.

The condition may commence with an embolus in the spleen, kidney or brain, the presenting signs in one case being optic neuritis with papillœdema. The symptoms, of course, would include sudden pain in the splenic or renal area, painful or sometimes painless hæmaturia, or nervous manifestations. The last are important, because in about 20% of cases the picture is predominantly neurological, aphasia or hemiplegia being not unusual complications. Embolic phenomena in the skin and subcutaneous tissues lead to ephemeral crops of petechiæ, which are sometimes painful, and Osler's nodes. Petechiæ should be looked for particularly in the cervical, clavicular and axillary regions. Clubbing of the fingers differs in this condition from most other types, in that the area is red and hot instead of blue and cold. In a small proportion of proven cases attempts at blood culture have been adamant in their failure; but in the majority special care and technique will produce the infecting organism, which in 95% of cases is the *Streptococcus viridans*. Cultures from the bone marrow occasionally produce results in resistant cases even while penicillin is being given.

What I wish to stress most emphatically, however, is the great need for early diagnosis. Why this urgency? Let us consider the results of the disease in the absence of penicillin: 32% of patients die from toxæmia; 29% die from cardiac failure; 25% die from emboli.

The heart is not enlarged early in the piece; but the combined effect of the original valvular heart disease, the further damage to the valves by the vegetative process, and the involvement of the myocardium by emboli, usually small, and Bracht-Wächter bodies, eventually leads to cardiac enlargement and failure in the majority of cases.

Here we can cure the infection with penicillin, but to what end? For we leave a dying or crippled heart.

Therefore I urge you, I even implore you, to make the diagnosis as early as possible, and to institute adequate treatment forthwith. One cannot always wait for the four main criteria to be obvious, and so any three of the four may be accepted as satisfactory.

Valvular heart disease, together with a positive blood finding is not sufficient, for after an abscessed tooth is chewed on or extracted, *Streptococcus viridans* is almost always put into circulation, but this does not necessarily mean that it will settle and multiply on the diseased valve. However, there sometimes comes a time when one cannot prove one's diagnosis, and one has to take a chance and treat the patient as suffering from bacterial endocarditis lest further delay should bring about cardiac failure.

Now I have presented to you merely a few main branches on a diagnostic tree, rather heavily pruned at that, and I leave it to my colleagues in the discussion to embellish it with the blossoms of their experience. My main object is to set a therapeutic course to guide the practitioner dealing with the average case.

Treatment.

Treatment is of two types, prophylactic and curative. The former, if successful, is easily the more important.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 30, 1948.

Prophylactic Treatment.

Prophylactic treatment involves suspecting all subjects of valvular heart disease and congenital heart conditions of being potential bacterial endocarditics, and of warning them to report any pyrexia, progressive malaise with loss of weight or possible embolic phenomena. If dental sepsis occurs, extractions should be made under a penicillin cover of 60,000 units every three hours for two days before and two days after the operation. This applies also to nose and throat operations.

Curative Treatment.

Curative treatment is comprised of general and special measures. Included under the former heading are adequate diet and rest in bed; it must always be remembered, however, that thrombophlebitis is a recognized complication of this disease, so that one must guard against excessive immobility. Vitamins are a necessary part of the diet, and may be given in additional doses according to one's susceptibility to the over-abundant literature on the subject. The history of the pre-penicillin days makes gloomy reading. Vaccines, sera, drugs of many types, fever therapy, blood transfusions, all proved of no real value. Intravenously administered antiseptics were tried. In fact, it is recorded that Boyd gave a solution of eusol intravenously; but the patient complained of pains and became unconscious, so the treatment was stopped. Surely under these circumstances the name eusol is hardly a happy choice for the solution. Then a candle-like glimmer of light appeared in the form of the sulphonamides. These were used alone or in combination with heparin or fever therapy, but the number of cures was disappointingly few. A few years ago Touroff cured a number of patients by ligating the infected patent *ductus arteriosus*, but this group is only a small section of the whole.

Penicillin then rose like the sun, its light at first being dimmed by the morning clouds of inadequate dosage; but now at its zenith it has searched out nearly all the dark haunts of the *Streptococcus viridans* and is capable of curing the infection in the vast majority of cases. It was first given intravenously, but caused trouble with the veins, so heparin was added. This adjunct is now rarely employed, owing to several disadvantages: (i) it may cause pyrexial reactions; (ii) it causes fragmentation of the vegetations, so leading to embolic accidents; (iii) it may cause hæmorrhage, especially into an area of cerebral softening due to an old infarct.

In order to increase the concentration of penicillin in the serum, para-amino hippuric acid, which by substrate competition interferes with excretion of the drug through the kidneys, was given intravenously; but this has now been virtually abandoned owing to the need for giving large doses intravenously.

Penicillin given orally is too wasteful and uncertain at present, but I feel that with some adjunct it has a future.

In order to avoid the necessity for frequent injections, penicillin in peanut oil and beeswax has been used; but the following are definite objections to its use: (i) it is wasteful, as much of the drug is destroyed by the tissues; (ii) it is uncertain in its action, for it is impossible to predict what serum penicillin level will be obtained, as this varies, not only from patient to patient, but even from day to day; (iii) it is a foreign body, and so produces a foreign body reaction; (iv) it may cause abscesses, into which further injections may be lost; (v) its unreliability is such as to render the more certain methods of administration advisable.

So we come to the present method of choice, intramuscular injection. This has been employed either continuously or at regular intervals, but the latter is usually the method of election. As I am concerned only with the average case, I can say that a suitable standard course is recognized as being the administration of 500,000 units per twenty-four hours given by three-hourly injections and maintained for twenty-eight days.

A new drug, "Caronamide", appears to have a future in that it delays the rapid renal excretion of penicillin. It has the advantage of being suitable for oral administration, and is capable of greatly increasing the serum penicillin concentration. In the majority of cases there is no synergistic action of sulphonamides when given in conjunction with penicillin. Inevitably in some cases in which penicillin has failed, streptomycin in doses of 1·0 to 1·5 grammes daily has been tried, and in some instances it has proved successful. Laboratory cooperation is necessary in the treatment of this condition, especially in the resistant cases, when the closest cooperation between physician and pathologist is *sine qua non*.

At the onset the pathologist will be looked to for the blood culture and if possible identification of the strain of organism, for some organisms, such as the alleged *Streptococcus viridans* S.B.E., require larger than normal doses of penicillin.

The pathologist can also estimate both the resistance of the organism to penicillin, and the serum penicillin level during treatment; but this latter is not usually essential, for clinical examination will generally demonstrate the efficacy of the treatment.

At this stage let me once again emphasize the necessity for early diagnosis.

In the case in which one cannot prove the diagnosis, but realizes the necessity for therapeutic action, one must make up one's mind that treatment is necessary and go straight ahead. Do not toy with it. Do not give a few days' penicillin treatment as a try-out. Once you decide to use the drug, give the full course and use no half measures; not only will such measures lead to relapses, but it has been proved in some cases that the resistance of the organism may be thereby greatly enhanced.

How are we to determine whether our measures are having a satisfactory effect? Perhaps we should consider the favourable indications first. After one week's penicillin treatment we expect to find the temperature normal, the blood sterile, and the patient possessed of a sense of well-being. These indications are favourable, but they do not necessarily mean that the infection is cured, for although there may be no circulating organisms, the vegetations probably still harbour nests of streptococci below the surface. Frequently the temperature rises during the second week, and it may remain elevated until the course is completed. This is no cause for concern, for it is a common result of penicillin therapy. As in all cases of cardiac disability, the pulse rate must be watched. It may be some three weeks after the patient is apyrexial before the pulse rate returns to normal. The blood sedimentation rate may not fall to normal for some five weeks after treatment is completed.

The hæmoglobin value and patient's weight usually begin to increase at the end of the course. In spite of all this we must always bear in mind the fact that the vegetations may not be well on the road to healing for at least three months. The unfavourable signs include the following. Blood culture may continue to give positive findings, or the temperature may not fall; in this case the dosage must be rapidly stepped up to 2,000,000 units daily, and the course lengthened to fifty-six days. A rise in the leucocyte count or in the blood sedimentation rate is a definite indication that all is not well. These two findings are thought to be reliable guides as to progress. Patients with electrocardiographic abnormalities have a less favourable prognosis than those with normal tracings.

One must remember that even while penicillin is acting well, the patient may die of toxæmia, heart failure, uræmia or embolism. It is only by early diagnosis that we can obviate such accidents. Later, after cure of the infection, death from cerebral hæmorrhage may occur—either hæmorrhage into a softened infarct, or hæmorrhage from rupture of a mycotic aneurysm.

A major embolus at this stage is rare. With the healing of the valvular lesions fibrosis may cause increasing disability—as, for example, by mitral stenosis—and so lead to cardiac enlargement and failure.

Complications.

Some of the complications which may appear during treatment are the following. (i) Every now and then

one is confronted with a sudden attack of abdominal pain with tenderness, rigidity and distension. If penicillin treatment is stopped for a few days, the pain disappears and treatment may be resumed without further trouble. (ii) Embolus may occur. (iii) Urticaria sometimes appears during the third week, and may persist so long as penicillin is being administered. (iv) Thrombophlebitis may occur.

Relapse may take place, but it is usually within a few weeks of the cessation of treatment. Fortunately it usually responds to treatment, though it may be necessary to give a higher dosage, perhaps 2,000,000 units daily for two months. Because of the possibility of relapse, there is some indication for periodic cultural examination of the blood during convalescence.

A late "relapse" is probably a reinfection. The question arises in regard to patients with a patent *ductus arteriosus* whether to treat them medically or surgically. All things being equal, it is probably wise to treat them in the normal manner with penicillin in order to cure the infection before ligating the ductus.

In special cases—for example, very resistant ones, cases of long standing, or cases in which the heart is beginning to cause concern—surgery may become a more urgent matter. When the condition is complicated by dental sepsis, it seems advisable to remove the offending teeth while the patient is still receiving penicillin.

Care during Convalescence.

It has been pointed out that the healing process in the valves takes more than three months to be completed. During this period further valvular interference with cardiac function may develop, owing to the fibrosis. It is, therefore, important to increase the patient's activity very slowly and gradually during convalescence, to avoid precipitating cardiac failure. The patient should not return to his normal work for six months.

Conclusion.

In conclusion, it is clear that failure to achieve success in the treatment of subacute bacterial endocarditis depends on a number of factors, including the following: (i) late diagnosis; (ii) inadequate dosage of penicillin; (iii) injections at intervals that are too long; (iv) too short a course of injections; (v) extreme resistance to penicillin of the infecting organism.

I am well aware that time has precluded me from mentioning many interesting details connected with my subject. It is also obvious that future progress will undoubtedly bring about some changes, and we trust some improvements; but like the law of the Medes and Persians, which altereth not, there are two things which will remain: (i) the urgent necessity for early diagnosis, and (ii) the vital importance of adequate treatment and control of convalescence.

Reviews.

FOOD AND DIETETICS.

THE tenth edition of "Hutchison's Food and Dietetics" follows the general plan adopted in the previous edition by its revisers, V. H. Mottram and G. Graham.[1] The book is now divided into four parts, "Diet in Normal Life", in which the general principles of the subject are discussed at some length, "The Nature of Foods", "Feeding in Infancy and Childhood", and "Diet in the Treatment of Disease". Dr. Hutchison's historical introduction to the subject, first published in 1934, is included without modification.

The main changes in this edition are in Part I, and are largely concerned with the variability of individual requirements as contrasted with average figures. The revisers appear to be rather obsessed with the fear that this aspect of nutrition may be regarded as susceptible to more scientific treatment than the data warrant. As the information which they cite is derived from observations and statements made by untrained subjects on their own diet and activity, the misgivings of the revisers are probably justified. The statements cited show enormous variation between Calorie intake and activity, for example. To conclude from this material, however, that a closer correlation between individual diet and activity does not exist accords to the data a weight which they do not merit. The revisers seem to have fallen into the error which they themselves deplore.

In addition to the discussion of the functions of the essential constituents of food, Part I contains a short chapter on the digestion, absorption, and metabolism of foods. Chapters on the processing, cooking and hygiene of food are also included.

In Part II the composition, properties, and use of a wide variety of foods, condiments, and beverages is dealt with. An attempt has been made to group the different types of foodstuff logically by considering them according to their importance as sources of energy, protein, minerals and vitamins. Otherwise the treatment of this part of the subject is of the same general character as in earlier editions. Where necessary the information has been brought up to date.

The part dealing with infant feeding, again contributed by Dr. C. H. Harris, has been rewritten. The discussion of diet in the treatment of disease has been considerably expanded. In it various special diets are given and discussed in detail. A section on intravenous feeding and the use of protein hydrolysates has been added. A comprehensive list of vitamin preparations, with their vitamin content, is included.

The book contains a wealth of information clearly presented. As much of this information could be obtained from other sources only with great difficulty, "Hutchison" remains an essential part of the library of anyone concerned with food and dietetics.

[1] "Hutchison's Food and the Principles of Dietetics", by V. H. Mottram, M.A. (Cantab.), and George Graham, M.D. (Cantab.), F.R.C.P. (London); Tenth Edition; 1948. London: Edward Arnold and Company. 8½" × 5½", pp. 754. Price: 21s.

SECTION CUTTING AND STAINING.

A SMALL, compact volume entitled "Practical Section Cutting and Staining" has been written by E. C. Clayden, senior technician in the morbid histology department of the Bland-Sutton Institute of Pathology, the Middlesex Hospital, London.[1] The methods and techniques described are stated to be those used in the institute. The author expresses his thanks to Dr. C. J. C. Britton and to members of the professorial staff of the Middlesex Hospital for help and criticism. The book, however, contains no endorsement or recommendation from the professorial staff of the Bland-Sutton Institute. Methods of preparation of paraffin sections, frozen sections and celloidin sections are presented clearly, simply and with admirable completeness. On the whole the book is conservative; for instance the Cambridge rocking microtome is described and illustrated, while the modern rotary microtome receives only a passing word and no mention is made of such labour-saving devices as the "autotechnicon".

essentially for technicians with little or no experience in the

In his preface the author states that this book is intended various methods of preparing routine sections, and expresses the hope that it will be found valuable for those intending to sit for the examinations of the Institute of Medical Laboratory Technique. As he states, most technicians acquire various useful practical "tips" and whenever possible these aids to better technique have been discussed in detail. The book contains 21 illustrations of a useful and practical nature.

[1] "Practical Section Cutting and Staining", by E. C. Clayden, F.I.M.L.T.; 1948. London: J. and A. Churchill, Limited. 5" × 8", pp. 140, with illustrations. Price: 9s.

ANÆSTHESIA.

A NEW EDITION of Hewer's "Recent Advances in Anæsthesia" is always a landmark for the professional anæsthetist. In this sixth edition[2] the author maintains his usual high standard and deals comprehensively with the many advances which have taken place in the four years since the previous edition.

The plan followed has been to preserve the framework of the text, but to insert passages to describe recent develop-

[2] "Recent Advances in Anæsthesia and Analgesia" (Including Oxygen Therapy), by C. Langton Hewer, M.B., B.S. (London), M.R.C.P. (London), D.A. (England); Sixth Edition; 1948. London: J. and A. Churchill, Limited. 8½" × 5", pp. 390, with illustrations. Price: 21s.

ments, such as local infiltration with a solution of procaine after the extravenous injection of thiopentone or the possibility in refrigerational analgesia of gas-gangrene infection from the ice. These insertions are made so deftly that the continuity of the text does not suffer. Important advances receive a whole new section, as with fractional spinal analgesia, or even a new chapter, as with the muscular relaxants. Despite this new matter, the size of the volume has been kept to within forty pages of the previous edition by masterly condensation and pruning.

The principal addition is the chapter upon relaxants. It is a valuable chapter, although certain aspects of the botany and pharmacology of "the curares", described in McIntyre's monograph, are not included. It is probable that Dr. Hewer's book was already in the press when McIntyre's work appeared. It is noteworthy that Dr. Hewer has discarded all attempts to estimate the dosage of tubocurarine from a basis of bodily weight. It seems to be agreed amongst anæsthetists in this country that *d*-tubocurarine chloride in a dose of 1·5 milligrammes per stone of body weight will fairly consistently abolish intercostal respiration and glottic activity, while a dose of 2·0 milligrammes per stone will inhibit the diaphragm as well. It is interesting, too, to read that Dr. Hewer does not insist upon tracheal intubation. It is a common practice in this country when curare is used, not as a precaution against possible bronchial spasm, but merely to prevent respiratory obstruction when tone is lost in the muscles of the tongue, pharynx and glottis.

The section upon the physical principles of gas flowmeters has been rewritten and improved. More attention is paid than formerly to the internal structure and principles of operation, as opposed to the external appearance, of apparatus. The section upon carbon dioxide absorption has been rewritten. The author repeats the warning, given universally until recently, against the possibility of causing alkalosis by over-ventilation of the lungs. It now seems, from the work of Gillespie and others, that alkalosis is in fact unlikely to be produced in this way. It follows that the user of apnœic methods of anæsthesia should be urged to concentrate upon adequate ventilation of the lungs, without much fear of producing alkalosis. The dangers of hypoventilation are so real, especially when curare is in question, that those of a somewhat theoretical hyperventilation become comparatively trivial.

Despite its conciseness, the book is encyclopædic in range. New facts are supplied upon topics so varied as the causation of surgical emphysema, the pharmacology of the barbiturates and the electrocardiographic changes seen under anæsthesia. Electronarcosis is discussed and many new data are given upon meningeal and nervous sequelæ of spinal analgesia. Intravenous analgesia with procaine receives due mention.

The sections upon anæsthesia for abdominal, thoracic and cardiac surgery have been brought up to date, although the last-mentioned does not describe the management of cases of the tetralogy of Fallot. A brief account is given of the apparatus developed in Sweden and Denmark for the mechanical performance of "controlled respiration". The reader is continually struck by *obiter dicta* of practical utility. Attention is drawn, for example, to the annoying bleeding which occurs at thyreoidectomy if the patient has been treated recently with thiouracil. Again, it is pointed out that, where paraldehyde is used as a basal narcotic, the subsequent anæsthesia should not be of the absorption type; the normal elimination of the narcotic through the lungs will be prevented and overdosage may result.

On the debit side, the section upon cyclopropane has been allowed to stand as in the previous edition. No reference is made to the important work of Dripps upon "cyclopropane shock" and emergence-delirium. The inference from that work is the fact that, in deep anæsthesia with cyclopropane, the patient's respiratory minute-volume becomes depressed to the point at which a sufficient amount of oxgyen can be taken into the body, but adequate elimination of carbon dioxide fails to occur. It follows that, in anæsthesia of this type, the patient's respiration must be manually "assisted" or even "controlled". This point is surely deserving of inclusion.

In successive editions the author has pruned away outdated material. One could wish that he had carried this process even further than he has done. For example, the use of partial rebreathing and of a minute-volume of delivery of five litres is surely outmoded. The same comment applies to certain references to carbon dioxide. When the author gives his own views, he is always sound, as in his warning against attempts to use carbon dioxide to stimulate the depressed respirations of "high" spinal analgesia. When he describes traditional practice, he is sometimes uncritical. The indications for carbon dioxide and oxygen mixtures, as opposed to oxygen, must be very few, whilst their potential dangers are real. It is time that their routine and indiscriminate use ceased in our teaching hospitals. Only thus can we rectify the present abuse of carbon dioxide in our smaller hospitals and nursing homes.

Yet again, Dr. Hewer advocates the adoption of Tredelenburg's posture in the treatment of circulatory depression. He should insert a warning that this posture throws a great burden upon the respiration of a handicapped patient. If used, it should be combined with the giving of oxygen and of manual "assistance" to the respiration. Finally, when describing abdominal operations under curare, the author gives a drawing of a patient in the attitude of "crucifixion", with arms held out on boards for intravenous drip infusion and blood pressure readings. This extreme abduction is unwise; it has been followed occasionally by stretch injury to the brachial plexus, whether under curare or otherwise.

The section upon oxygen therapy is complete, but one point might have been emphasized. Lung inflators and similar apparatus are very well in their way, but they are not always at hand and in working order in an emergency. The application to the patient's face of an ordinary gas mask and bag filled with oxygen, followed by rhythmic inflation of the lungs as in "controlled respiration", is as efficient as any mechanical form of resuscitation and is capable of immediate application.

In conclusion, the book retains the agreeable format, paper and binding of previous editions. There is even greater variety in the type and the proof-reading has been excellent. The author has covered the recent advances in anæsthesia most capably and has sustained his high reputation. At the same time, a warning to those unfamiliar with the earlier editions may not be misplaced. The author's method is to cite, with references, the views of hundreds of authorities. The inclusion of a particular view or method is hence no guarantee that the author approves of it. Sometimes he states his own views; frequently he does not. Since some of the methods cited are far from being orthodox, the principle *caveat lector* must apply. The reader would be most unwise who set out to employ a given method without first consulting the original paper and the comments upon it of other workers, to all of which numerical references are given. Dr. Hewer's aim is to produce a general review of modern anæsthesia for the stimulus of the professional anæsthetist. He has not set out to write a text-book, still less a technical manual.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Child in Health and Disease: A Textbook for Students and Practitioners of Medicine", by Clifford G. Grulee, M.D., and R. Cannon Eley, M.D.; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 6¾", pp. 1084, with many illustrations. Price: 90s.

The authors write for students and practitioners and claim that they deal with the practical application of medical investigations at the bedside.

"Technique of Treatment for the Cerebral Palsy Child", by Paula F. Egel; Introduction by Winthrop M. Phelps, M.D.; Appendix by Moir P. Tanner, F.A.C.H.A.; 1948. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Proprietary, Limited. 8½" × 5½", pp. 208, with 49 illustrations. Price: 26s. 6d.

The book presents the problems of the treatment of cerebral palsy from the point of view of one who actually carries out the treatment procedures recommended by the physician.

"The Medical Clinics of North America" (issued every two months); 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Philadelphia Number. 9" × 5¼", pp. 304, with illustrations. Price: £4 12s. 6d. (paper binding) and £5 10s. 6d. (cloth binding) per clinic year.

Comprises a symposium on recent advances in gynæcology and obstetrics.

"The Surgical Clinics of North America" (issued every two months); 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Nationwide Number. 9" × 5¼", pp. 286, with illustrations. Price: £4 12s. d. (paper binding) and £5 10s. (cloth binding) per clinic year.

The main subject is a symposium on gastro-intestinal surgery; there are also articles on general surgical subjects.

The Medical Journal of Australia

SATURDAY, MARCH 12, 1949.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE RETIREMENT OF SIR HENRY NEWLAND FROM THE FEDERAL COUNCIL.

THE abrupt termination by the Commonwealth Minister for Health, Senator N. E. McKenna, of discussions on a national health service between himself as representing the Commonwealth Government and the Federal Council of the British Medical Association in Australia as representing the medical profession of the country has precipitated an event which has been threatening for some considerable time. Sir Henry Newland has retired from membership of the Federal Council and the Council has lost its President. During recent years in the history of Australian medicine no man has given more devoted service to his profession than Sir Henry Newland, no man has shown more unswerving loyalty to the ideals of medicine and fixity of purpose in their attainment, and no man has more richly earned the right to seek relief from the cares of high office. The office of President of the Federal Council is no sinecure; it does not consist merely in the taking of the chair at meetings of the Council or even in the making of replies to pertinent and impertinent statements by public persons, many of whom are ill-informed. The volume of correspondence is very great and in the interval between meetings certain executive powers delegated by the Council have to be used. Not a little wisdom is needed for this task. Beyond this, of course, certain medical bodies not connected with the British Medical Association and scientific and other bodies outside the immediate boundaries of medicine make demands on the time of the President. The work of a medical president in an organization of such size as that embracing the six Branches of the British Medical Association in Australia is something like the task of a master mariner. The difference lies chiefly in the fact that the medical president is often expected to have powers of divination. The captain of a ship always has accurate charts of the seas he is sailing; he also has modern pieces of apparatus which give him depth soundings, he has wireless and other devices not suspected by the longshoreman—in fact some people would say that he was clever if he managed to pile up his ship on the rocks in the face of all his safeguards. The medical president has no accurate charts of currents, rocks and sandbanks. Some of these hazards are built up of the prejudices and purblindness of those around him and appear in unexpected places. Sometimes opportunism or some immediate passing benefit waits just ahead, like a Lorelei wielding her golden comb, and the medical ship of State will be damaged if not wrecked unless the captain has a sure eye and a single purpose. The passengers of a medical ship are quick to find fault unless they have learned to know and trust the master, and even members of the crew are not slow to murmur until they have learned confidence. When we realize that Sir Henry Newland has commanded the Australian ship of medicine since the year 1930 and that he has stepped down from the command with the entire confidence and esteem of every member of the crew, we begin to understand something of what he has done for the profession in this country.

That Sir Henry Newland's career has been singularly rich in achievement was made clear at a dinner given in his honour in Melbourne on March 1, 1949, by the members of the Federal Council, with the members of the Secretariat and the Editor of this journal. Dr. Victor Hurley, who has succeeded Sir Henry Newland as President of the Federal Council, presided at the dinner and proposed the toast of the guest of the evening. Nine others spoke in its support. The facts which emerged included first of all Sir Henry Newland's joining the Federal Committee in 1921. Of this body he became President in 1930, and when the Federal Committee became the Federal Council in 1933, Sir Henry Newland continued as President. In latter years he consulted his Vice-President before he accepted nomination each year to the presidency. The General Secretary has revealed that since 1944 Sir Henry Newland has wished to retire from the office of President. He is one of those, however, who believe that it is a mistake to change horses in mid-stream, and while the question of a national medical service was being actively discussed with the Government, he thought that it would have weakened the position of the Association had he surrendered his office. All who have been associated with him in the work of the Federal Council during these years have been amazed at his knowledge and understanding of the requirements and procedures of general practice as well as of those associated with practice in the several specialties. In matters affecting all forms of practice Sir Henry Newland was always able to take a long-range view when necessary and he based his decisions on the highest ethical plane. It was his insight into the needs of practitioner and patient which made him especially valuable as a member of the Central Medical Coordination Committee during the war years. This work has been little known by the rank and file of the profession. The delegates from South Australia spoke of Sir Henry Newland's ability as a surgeon and as a teacher, and referred to the deference paid by the South Australian Branch Council to his opinions. Sir Henry Newland's retirement from the Federal Council will mean his retirement from the South Australian Branch Council, unless some special means are found by which his membership of that body can be retained. Dr. W. F. Simmons in a singularly telling speech recounted Sir Henry Newland's achievements in the war of 1914-1918 in the field of surgery and declared that decorations received had never been more richly deserved. Dr. A. E. Lee, of Brisbane, reminded

Sir Henry Newland that he alone, of all those present, had been obliged on one occasion to move a motion of no confidence in him on account of something with which the Queensland Branch had disagreed, and he recalled the courtesy and large-mindedness with which this action had been received. The General Secretary gave those present an insight into the volume of work carried out by the President between the regular meetings of the Council.

In some quarters the suggestion has been made that Sir Henry Newland's retirement from the presidency of the Federal Council has some sinister significance. The suggestion is incorrect and mischievous. Sir Henry Newland's leadership has been successful—and, more than this, it has been an inspiration to his colleagues. The Federal Council has adopted the following resolution as a permanent record in its minutes:

> The Federal Council of the British Medical Association in Australia at its meeting in Melbourne on March 1, 1949, wishes to place on record, on the occasion of his retirement as President, its appreciation of and its gratitude to Sir Henry Simpson Newland for his unceasing labours during many years of service. Coming to the Federal Committee as a representative of the South Australian Branch in 1921, Sir Henry Newland at once took a prominent part in its deliberations and in 1930 was elected President. When in 1933 the Federal Committee became the Federal Council, Sir Henry continued in the office of President and has served with distinction since that time. During the difficult years of war his interest did not cease nor his energy falter. As a leader he has set a high standard and the Council knows that his influence will extend far into the future. He has the affectionate regard of every member.

Sir Henry Newland is a Vice-President of the Parent Association in England; he has received the gold medal of the British Medical Association in Australia, and his portrait has been presented to him by the South Australian Branch. He has received the honour of knighthood from His Majesty the King. If he had an opportunity to express his opinion, Sir Henry Newland would in all probability not wish for further honour to be done him—he is that kind of man. He may rest assured, however, that the words of the Federal Council's minute will be re-echoed throughout the length and breadth of Australia.

Current Comment.

EXPERIMENTAL SALMONELLA INFECTIONS IN AUSTRALIAN COCKROACHES.

THE cockroach is commonly regarded as a "dirty" creature and treated with disfavour. However, it abounds in many parts of Australia and is, in general, tolerated, perhaps because of a prevalent vagueness regarding its role as a carrier of disease. I. M. Mackerras and P. Pope[1] point out that the occurrence of naturally infected cockroaches during an epidemic in Brisbane has been previously reported, and various workers have recorded the experimental infection of cockroaches with bacteria, but few of the experiments were carried on for more than three days and none seemed to demonstrate that the organisms did more than pass alive through the intestinal tract of the insect. Mackerras and Pope have investigated the position to a more conclusive stage. The cockroaches were mounted on their backs on blocks of hard paraffin, so that their droppings could be caught on a petri dish containing an appropriate medium. They were fed on sterile sucrose solution and bread. With suitable precautions and control arrangements, the insects were fed with Salmonellas and the results observed for quite long periods. It was found that the cockroaches acquired infections readily, about 75% of the insects becoming infected, and about 15% became "chronic carriers", in that they retained the infection for more than a fortnight. They appeared to be intermittent excretors of the organism, but it was found that viable Salmonellas were excreted in the fæces up to forty days from infection. Mackerras and Pope point out that, as cockroaches feed readily on both fæces and food, their capacity to spread salmonellosis is thus by no means negligible. During a recent outbreak of gastro-enteritis in Brisbane, Salmonellas were isolated from ward sinks and brushes, to which cockroaches would have ready access, and from cockroaches captured in the wards. It was not thought likely that they would infect the food of infants, but they could easily have contaminated the food of older children and adults and thus contributed to the vicious circle of infection. Their exact role in any particular case, as Mackerras and Pope state, can be determined only by detailed epidemiological studies, but these investigators, it must be agreed, have adduced sufficient evidence to justify intensive control measures in any area where cockroaches and Salmonella infections are prevalent. Indeed, a continuous public and private war on the cockroach in all circumstances would seem a worthy measure.

[1] *The Australian Journal of Experimental Biology and Medical Science*, November, 1948.

THE AROMATIC DIAMIDINE DRUGS.

ATTENTION has been drawn in these pages to trials of drugs of the aromatic diamidine series in various conditions in which the hope was raised of altering perversions of cell metabolism, such as are found in neoplasms. This general statement in no sense expresses the highly specialized nature of research into the actions and reactions of the living and breathing unit, the cell, or into the molecular structure of substances, powerful yet safe in the treatment of disease. It is usually a long time before basic research pays a dividend at the bedside, but it is interesting to review the stages between, beset by patient toil, before we have "the extract, flasked and fine, and priced and salable at last". An interesting review has been published by E. B. Schoenbach and E. M. Greenspan from the Johns Hopkins University School of Medicine tracing the work of the last ten years and more on stilbamidine, pentamidine, propamidine and other drugs of this series.[1] This research is connected with the name of Warrington Yorke perhaps more closely than with that of any other among the long list of workers who have brought knowledge to its present point of achievement and promise. Yorke and others perceived a fallacy in some of the early work in looking for an anti-trypanocidal agent. The delicacy of the glucose metabolism of trypanosomes led to the trial of hypoglycæmic agents, and the activity of a guanidine derivative, "Synthalin", in mice and rats infected with resistant strains of trypanosomes at first suggested that the key to the attack on this organism would be found in interference with sugar metabolism. Yorke and his colleagues, however, showed that "Synthalin", formerly tried in diabetes, but found too toxic for use, was trypanocidal in too low a dilution for any hypoglycæmic action to be effective, and with real insight, postulated a specific drug action. Thence started one of those long searches after a connexion between molecular structure and specific action, brought to fruition in the successful clinical use of the aromatic diamidines in human trypanosomiasis and leishmaniasis. The details of the structural researches on these drugs are too technical for the average practitioner, even if he had time to devote to the subject; but it was perhaps characteristic of the curious beginnings of such work that the group originally thought to be of prime importance, the diguanidines, was soon discarded for the diamidines. A search for less toxic products revealed the fundamental chemotherapeutic constituent, and once the terminal basic group was established variations in the carrier chains could be made without loss in potency. The

[1] *Medicine*, September, 1948.

mode of action was investigated; in the course of this work the striking specific race differences became obvious in different strains of trypanosomes. But not only these protozoal organisms were studied, for it was found that as the length of the chains increased so the compounds became more active against bacteria also. Propamidine in particular was found to be very active against Gram-positive cocci even in the presence of para-aminobenzoic acid, unlike the sulphonamides. In fact, the components of the *B* vitamin group had no anti-diamidine activity. Some analogy seems to exist between certain features of the action of these drugs and the activity of the cationic detergents which have been introduced for surgical and domestic purposes.

At this point the story begins a new chapter. Dale suggested that since the diamidines inhibited oxidation in bacterial and protozoal cells, they might have similar properties in the cells of normal animal tissue, and perhaps even in those showing disturbed and irregular metabolism, like the cells of neoplasms. Though toxic action has hindered some of the animal experiments, inhibition of tumour cells *in vitro* has been proved, and denaturation of nucleoprotein has also been studied. This may seem rather academic at first, but in the work of Snapper and others on the effect of stilbamidine in multiple myeloma there is clear evidence of the action of the drug on the myeloma cells, particularly in the finding that 80% of these cells develop basophile inclusion bodies. Further studies on these granules are proceeding, and if they confirm the supposition that specific morphological changes have been produced in myeloma cells by a drug given by mouth in the ordinary way, a new milestone in medicine will have been passed. This work and other examples of similar observations have been the subject of comment here before, and it is really important to try to look beyond the immediately promising clinical results and see the principle involved. It is not unfair to introduce a touch of pessimism and to remark that it is likely that many disappointments will be encountered, but new fields are opening in biological research with the use of this weapon of the systematic investigation of compounds related by the possession of a specific basic factor. There are some curious features about these drugs. For example, although stilbamidine in dilute solutions fluoresces brilliantly in ultra-violet light, and has a narrow absorption band in the spectrum, no simple accurate method has yet been devised for its quantitative estimation owing to its adsorption to proteins. A great deal of work has also been carried out on the pharmacological and toxic properties of these drugs. Work is actively proceeding on the clinical applications in human trypanosomiasis, which, in view of the continuous contraction of the time dimension of our world, is now of great practical importance. Recent researches promise even greater advances. Even better known is the value of the aromatic diamidines in human leishmaniasis, though the most potent preparation, stilbamidine, is perhaps too toxic for general use. Whether the application of this drug to the problem of myeloma and other diseases of the blood associated with abnormal cell development will be fruitful cannot yet be told. Admittedly, some of the clinical trials have been made on a purely empirical basis, but it is encouraging to review the remarkable progress that has been made in producing these drugs and in gaining insight into their mode of action on animal tissue cells. It is not impossible that future research may reveal new agents effective not only in the attack on bacterial and non-bacterial infections, but perhaps, too, on neoplastic disease.

"Q" FEVER IN GREAT BRITAIN.

THE history of "Q" fever was reviewed in these columns on July 26, 1947, starting from the first description of the disease by Derrick in 1937 and the isolation of the causal rickettsial organism by Burnet. Particular reference was made to an outbreak in Amarillo, a city in the State of Texas, of a disease whose clinical features differed little from those described by Derrick; in two cases a rickettsial agent was isolated apparently identical with *Rickettsia burneti* and in the majority of cases the diagnosis was confirmed serologically. It was emphasized by the investigators of the Amarillo outbreak that serological tests were preferable to attempts to isolate *Rickettsia burneti* because of the extreme infectiousness of this agent in the laboratory. The results indicated that the complement-fixation test was both sensitive and specific, though mainly of use for retrospective diagnosis as the titres reached their highest levels in the later stages of the disease and during convalescence. A further comment was that "Q" fever must be considered in the investigation of atypical and non-bacterial pneumonia and undiagnosed febrile illnesses. However, it is only gradually being realized that this disease is far more widespread than the original reports might have suggested. Outbreaks occurred amongst British and American troops in Italy in 1946 and cases have been reported from Germany, Switzerland and Turkey. The latest report comes from Great Britain, where M. G. P. Stoker[1] has been investigating cases of atypical pneumonia and similar acute respiratory illnesses. Of 24 sporadic cases of atypical pneumonia so far investigated "Q"-fever complement-fixing antibodies were found in the sera in three. One of the patients had spent six months in Germany as a prisoner during the war, so that it was not possible to prove that the antibodies were not the result of a mild attack while he was in Germany; the evidence was, however, strong that he had contracted the relevant illness in Great Britain. The other two patients had never been out of the country, so that the evidence, though limited, suggests that "Q" fever occurs in Great Britain. More important still it emphasizes the need to bear "Q" fever in mind in the investigation of atypical pneumonia, acute respiratory illness and undiagnosed febrile states.

SUCTION APPLIED TO APPENDICECTOMY.

THE friable, acutely inflamed appendix may worry the most experienced surgeons; the results of its mishandling may be serious even with the means now available of combating infection. John Devine[2] points out that the danger of infecting the wound during appendicectomy is greatest during the delivery of the appendix through the wound, and that any method aimed at protecting the wound must be applicable to the appendix while it is still inside the abdomen. This is an important drawback, as Devine explains, to the suggestion of B. J. Ficarra[3] that the appendix should be encased in a rubber finger cot after it has been liberated from its mesenteric attachment; protection is provided against the effects of perforation during manipulation, but only after the appendix has been delivered through the wound. Devine suggests a method of isolating the appendix which starts as soon as it is exposed in the abdomen. A tube of suitable internal diameter is attached by means of fine pressure tubing to the ordinary operating-room source of suction; as soon as the tip (or base when the appendix is removed in a retrograde manner) presents, the tube is applied to it and the appendix is gently sucked into the tube. As the meso-appendix is divided, more of the appendix is sucked into the tube. The tube actually aids in the removal of the appendix and may, for example, simplify the separation of the tip of an appendix from adhesions in retrograde removal, being forced by the suction along the correct planes with little interference from the surgeon. Retrograde removal is made much easier and in many cases more desirable. All the time the suction tube is isolating the infected appendix from the wound and peritoneal cavity and is able to deal immediately with spilt pus or pieces of friable tissue. After trying various materials Devine has found a glass tube the most satisfactory; for standard use it is straight, but a curve at the end helps if the base of the appendix has to be tied deep in the wound. He has only used the tube in something over twenty cases, so can present no statistical study of its effect on the incidence of wound infection, but the idea commends itself.

[1] *The Lancet*, January 29, 1949.
[2] *The Lancet*, February 5, 1949.
[3] *The American Journal of Surgery*, August, 1945.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Technique of Iridencleisis.

L. WEEKERS AND R. WEEKERS (*The British Journal of Ophthalmology*, December, 1948) describe a technique of iridencleisis which they have used in hundreds of cases over the past eighteen years. The success of the operation depends on a spontaneous prolapse of the iris. The use of miotics is discontinued the evening before operation. A conjunctival flap is dissected down to the limbus from the 10 o'clock around to the 2 o'clock position, and when completed should drape over the cornea spontaneously. An incision is made through the sclera just within the limbus at the 12 o'clock position, the incision being completed with one bold stroke of the knife. A spontaneous prolapse of iris occurs. The summit of the iris prolapse is grasped with two iris forceps, one on each side, and a slight horizontal pull then tears the iris into two fragments. They are merely separated from each other. The conjunctival flap is sutured into position, penicillin drops are instilled and a uniocular dressing is applied. The dressing is removed after forty-eight hours, being reapplied at night for an additional five days. Atropine is instilled twice daily for two days as soon as the anterior chamber is formed, which is usually within two or three days.

Surgery of Congenital Cataracts.

FREDERICK C. CORDES (*American Journal of Ophthalmology*, September, 1948) considers that for congenital cataract the first eye should be operated upon at the age of six months if the cataract is sufficiently dense to interfere with fixation; the second eye is operated upon between the second and third years. When the lens opacity is sufficient to interfere with the possibility of the child's learning to read, the cataract should be removed before the school years. The prognosis as to the amount of post-operative vision must be guarded. The ideal seems to be that a single post-operative procedure should provide for the removal of at least the major portion of the lens so as to permit the remainder to be absorbed without further surgical interference. In addition there should be no interference with the pupil and a minimum danger of iris incarceration or adherence to the operative wound. The most universally employed operation is simple discission. In the author's opinion the disadvantage is that the operation has to be repeated and, although complications are rare, they include cessation of absorption, post-operative low-grade and prolonged irido-cyclitis, and organization of capsular tags and remnants into a dense membrane. Secondary glaucoma also occurs. Various modifications of the simple discission may be employed. The author favours what he calls a modified Barkan operation for all cases except certain types of congenital cataract. He describes this operation in detail. He states that as other anomalies are often present the prognosis should be guarded. The embryonal nuclear cataract causes so little interference with vision that surgery is not indicated. Zonular (lamellar) cataract is usually associated with other congenital defects of the eye so that the operative prognosis is not good. Membranous cataract responds well to discission. The disk-shaped cataract, which consists of two types of cataract in the one individual, that is, a membranous central area and a soft cataract in the periphery, is best treated by discission followed by attack on the membrane with de Wecker scissors. For rubella cataract the author uses the linear extraction method, as in many instances the very dense central portion of the lens has proved resistant to the needle. For retrolental fibroplasia the procedure of Reese and Payne is recommended, namely, discission of the lens and finally the making of a vertical cut in the retrolental fibrous tissue with de Wecker's scissors. Cataracts following intrauterine inflammation have a very bad prognosis, and operative interference is invariably followed by inflammation and fibrosis.

Placental Extract for Herpetic Keratitis and Superficial Punctate Keratitis.

BERNARD KRONENBERG (*American Journal of Ophthalmology*, September, 1948) presents the results of the work done with the various placental extracts in the treatment of a variety of ocular diseases. The technique of the preparation of three placental extracts is described. In six cases superficial punctate keratitis and herpetic keratitis were healed with an aqueous extract with gratifying results. The dosage used was two millilitres of concentrated solution given intramuscularly. These patients had previously failed to respond to local therapy, foreign protein therapy or administration of vitamins for over one month. In two cases herpetic keratitis which responded to this treatment had failed to respond to any local therapy. In the treatment of *retinitis pigmentosa* the author was unable to confirm Filatov's satisfactory results. He states that the placental tissue coagulant may have its uses before operation to lower blood-clotting time in certain patients, such as diabetics.

Anterior Flap Sclerotomy with Basal Iridencleisis.

H. B. STALLARD (*The British Journal of Ophthalmology*, October, 1948) describes the technique of a combined operation for glaucoma. The operation consists in the reflection of a conjunctival flap, the fashioning of a scleral flap hinged on the corneo-scleral junction, a limited cyclodialysis and the inclusion of a basal tongue of iris between the lips of the sclera, the *sphincter pupillæ* being left intact. He has used the procedure in 29 cases of chronic glaucoma and two of acute congestive glaucoma.

Failure of Operation for Chronic Simple Glaucoma.

B. F. PAYNE (*American Journal of Ophthalmology*, August, 1948) has studied histologically approximately 100 eyes which had been operated upon for glaucoma at the New York Eye and Ear Infirmary. They all showed at least three characteristics: (i) adhesions between iris and cornea; (ii) closure of new filtration channels with fibrosis; (iii) inflammation of the uvea. The eyes which were examined had been subjected to iridectomy, iridencleisis and trephination. In those in which iridectomy had failed histological examination showed that the normal iris angle was not restored and the adhesions were not relieved. After an unsuccessful iris inclusion operation atrophic uveal tissue was found in the operative wound. In a typical case of failure after trephine there was closure of the operative wound by dense connective tissue containing fragments of pigment, lymphocytes and new blood vessels; the lens was in close relation to the filtration wound. These two factors, the authors state, prevent a successful result.

Scleral Resection for Retinal Detachment.

SEYMOUR PHILPS (*The British Journal of Ophthalmology*, November, 1948) presents a preliminary report on scleral resection for retinal detachment. The indications for this operation are: (i) detachments with multiple tears and thin atrophic retina when diathermy has failed, or when the retina breaks away extensively after a successful diathermy operation; (ii) detachment when no rent can be found because of obstruction to retinal examination; (iii) long-standing detachments when the retina has shrunk and will no longer fit the globe; (iv) some detachments in very high myopia when the vitreous retraction and thin retina gave little chance of success with diathermy. The scleral resection shortens the eye, and this allows the shrunken retina once more to fit the globe; it collapses the peripheral retina which contains the rents or degenerative patches which have caused the detachment. It may be employed round the whole 360° of the globe, though not more than 180° should be attempted at a single operation and the centre of the scleral arc which is resected should be over the probable site of the retinal hole. The technique, which is not difficult, is described in detail and follows closely the operation which was previously described by Vail in 1946.

"Antistine" and Ocular Allergy.

PAUL HURWITZ (*American Journal of Ophthalmology*, November, 1948) discusses the use of the antihistamine preparation "Antistine" for allergic conditions of the eye. He states that allergy may affect any portion of the eye or adnexa and is usually associated with allergic conditions elsewhere in the body, especially with allergic rhinitis and gastro-intestinal disorders. The allergic reaction in the eye is characterized by spasm of smooth muscle, capillary dilatation and increased permeability. The allergic reaction is due to the liberation of "H" substance or histamine. Antihistamine agents are presumed to be effective through neutralization of the "H" substance or through replacement of the space in the cell that might be occupied by histamine. Pharmacologically it has been found that "Antistine" has a very low toxicity, does not accumulate rapidly and has strong antihistaminic activity. The author used the drug in fifty cases of ocular allergy associated with hay fever, allergic states other than hay fever, vernal conjunctivitis and palpebral urticaria, dermatitis and angioneurotic œdema. In nearly every case there was relief

of symptoms, although the basic pathological state was not altered. The dosage required to maintain relief varied from one drop of "Antistine" solution every three or four hours to one drop daily and in some cases one drop every second day.

OTO-RHINO-LARYNGOLOGY.

The Loudness Recruitment Phenomenon.

R. M. DIX, C. S. HALLPIKE AND J. D. HOOD (*The Journal of Laryngology and Otology*, November, 1948) state that clinical studies to elucidate further the significance of the loudness recruitment phenomenon described by Fowler in 1936 have been carried out by the otological research unit at the National Hospital, Queen Square, London. They point out that the relative deafness of the affected ear evident at threshold disappears at higher intensities of the test sound in certain forms of inner ear or nerve deafness. It is believed that as loudness is increased the more strongly activated hair cells will be sufficient to saturate the cochlear fibres or the cells of the cochlear nuclei, so that the cerebral cortex will receive the same number of impulses per second from both ears and will then perceive the tone delivered to the diseased ear as strongly as the tone delivered to the normal or less affected ear. In conduction deafness, such as that due to middle ear disease, the lesion apparently acts as an equal attenuator for all intensities of the test tone, so that the loudness recruitment phenomenon cannot then be demonstrated. The phenomenon is thus considered to be pathognomic of neural deafness. The present study included the observation of the form of this phenomenon in 30 cases of unilateral deafness due to Ménière's disease, and in 20 of degeneration of the eighth nerve due to neurofibroma or other space-occupying lesions of the cerebello-pontine angle. It is accepted that the actual lesion of Ménière's disease is one affecting the endolymph system of the cochlea and its contained end-organs. In this latter condition the cells of the spiral ganglion are said to appear quite normal in number and structure, while there is no apparent reduction in number of the cochlear nerve fibres in the modiolus or in the osseus spiral lamina. The organ of Corti has been shown to be greatly distorted and compressed in a number of case reports, but in many others it has appeared to be normal. It is suggested that the changes are perhaps transient and reversible and are due to gross distension of the endolymph system. In the majority of the 30 cases of Ménière's disease, deafness was substantially unilateral. In all 30 cases loudness recruitment was pronounced. There were 11 cases of neurofibroma of the eighth nerve and nine of other varieties of cerebello-pontine-angle lesions involving the eighth nerve. In many cases of this type the deafness of the affected ear may be too severe for application of the loudness balance tests. In 14 of the cases in this series a complete absence of recruitment was found, while in the remaining six only a slight amount of recruitment was evident. Thus, while loudness recruitment has been previously regarded as characteristic of nerve deafness, here is a form of nerve deafness in which recruitment is not present at all. It is suggested that in the loudness balance test we now have at our disposal a test procedure which should prove of great value in making clear the distinction between end-organ deafness and nerve-fibre deafness. Experimental electro-physiological data indicate that as stimulus intensity is increased there occurs a spatial spread within the cochlea with progressive activation of additional nerve fibres. In the case with a degenerated nerve there is only a limited survival of fibres and, a constant fibre survival rate being given, this would be compensated by a constant fractional increment of stimulus intensity at all points of the intensity scale, so that the loudness recruitment phenomenon might be expected to be absent, as clinically is found to be the case.

Movements in Larynx with Alteration in Pitch.

A. G. H. MITCHINSON AND J. M. JOFFEY (*Journal of Anatomy*, April, 1948) have investigated radiographically the movements of different parts of the larynx and related structures during alterations in the pitch of the voice. Twenty subjects, ten male and ten female, ranging in age from sixteen to fifty years, underwent X-ray examination during the humming of (*a*) low notes and (*b*) high notes. In changing from a low to a high note the vocal folds usually became elongated, and rotated. The larynx and hyoid bone were raised and the supraglottic space was shortened. In three subjects with considerable practice in singing, the elongation of the vocal folds was greater than in the remaining seventeen subjects.

The Functions of the Round Window.

E. G. WEVER AND M. LAWRENCE (*Annals of Otology, Rhinology and Laryngology*, September, 1948) have studied the function of the round window by immobilizating it in various ways while recording the electrical potentials of the cochlea. They state that the effects vary both with the method of immobilization and with the condition of the ear. When the ear was intact, and the sound stimulus was directed at the ear drum in the usual way, a partial immobilization of the round-window membrane by application of a prod to its surface had no measurable effect. Under the same conditions, the application of a tube over the window, the exertion of a heavy air pressure on it and the plugging of the niche with wax all had nearly the same effect, which was a progressive reduction of the response to high tones, amounting at the most to about ten decibels. When the middle ear was removed and the sounds were introduced through a tube sealed over the oval window, the same procedures had little or no effect on the responses to high tones, but in some ears they reduced the responses to low tones. Possibilities are considered for the explanation of these results. Check experiments seem to exclude a disturbance of the sound field as a result of the manipulations. Also ruled out is the possibility that the results can be accounted for as a modification of a secondary path of sound transmission by way of the tympanic space and the round window. Such transmission can have only negligible effects. It is concluded that the blocking effects the mechanical impedance of the ear. The authors point out that a surprising result is that all forms of blocking, whether partial or complete, cause only minor alterations of the sensitivity. The changes occur mostly at one end or the other of the frequency scale, and are usually of the order of five decibels, rarely exceeding ten decibels.

Surgical Treatment of Otosclerosis.

G. HERBERTS (*Upsala läkareförenings förhandlingar*, October, 1948) states that the earlier experimental operations for otosclerotic deafness included attempts at stapes extraction. For various reasons the operation was given up. In 1896 Passow obtained some good primary hearing results on making a new labyrinthine window in front of and below the oval window. Earlier methods had produced negative results probably due to mechanical or infectious injury of the labyrinth. The improvements in technique, especially lighting and the use of the operating microscope, as well as antibiotic therapy, lend support to the possibility of further attack on the stapes region. According to the author it is most important to keep the membrane covering the inside of the base of the stapes. Whereas this membrane is found to be very thin in normal cadavers, in cases of otosclerosis there is less difficulty in preserving it as it seems to be thickened. Two patients have been operated on. The approach is made through an endaural incision and partial mastoidectomy. The incus is taken away and a flap is made in the usual manner. With the microscope it is possible to work downward across the facial nerve so that the head of the stapes and adjacent structures are visible. When the *corda tympani* and stapedius tendon have been severed it is possible to determine whether the stapes is ankylosed. It was found that the otosclerotic focus made a bridge from the antero-inferior portion of the oval window to the adjacent segment of the stapes. The postero-inferior part of the stapes was free from the wall of the oval window recess. By loosening off the mucosa from the promontory immediately in front of the oval window and working thence upwards and downwards and backwards it was possible to by-pass the oval window, and by careful scraping to separate and extract the stapes from the window with the window membrane preserved. There was some quick-passing dizziness, and simultaneously a considerable improvement of hearing occurred. In the second case an attempt was made also to scrape away the bone corresponding to the supposed otosclerotic focus. The suspected bone was found to be of chalky consistency without being especially hard. In spite of great care the bone being curetted gave way and small drops of fluid escaped. The patient now registered a very strong hearing improvement. As a fistula to the vestibulum evidently had been obtained, the boundary of the window was enlarged and bone splinters were removed with the window membrane. The plastic flap from the tympanic membrane and meatus was able without difficulty to be made to cover the widened oval window. Hearing in both cases decreased after a few days and increased again the next. It is considered too early yet to judge the functional results of the operation.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 9, 1948, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. J. KEMPSON MADDOX, the President-Elect, in the chair.

Pulmonary Infarction and Anticoagulant Therapy.

DR. A. W. MORROW read a paper entitled "Pulmonary Infarction and Anticoagulant Therapy" (see page 341).

DR. H. G. CUMMINE read a paper entitled "Anticoagulant Therapy" (see page 342).

DR. G. V. RUDD said that both speakers had dealt with interesting aspects of the subject under discussion. Dr. Morrow's paper had given valuable information about some of the problems of the difficult diagnosis of pulmonary embolism and infarction. Dr. Rudd said that in addition to the rapidly fatal embolism there was a second kind of pulmonary embolism which might be called incidental pulmonary embolism. In that condition, as had been pointed out, only smaller arteries were involved and the affected area of lung was not infarcted but hyperæmic and œdematous. If enough small arterial branches were affected, the obstruction to the circulation might be sufficient to cause death. A third type was pulmonary embolism with infarction in which either the larger or the smaller branches were involved. If the patient should die in the first twenty-four or forty-eight hours (the period described by Dr. Morrow as that in which the radiographic appearances were uncertain), autopsy would reveal that the infarcted area was hæmorrhagic and blood oozed freely from the cut surface. Soon afterwards, however, during the stage in which the radiographic appearances became more precise, the infarct took on a firmer consistency, and at that stage the cut surface was dry. Organization of the infarct soon commenced and later destruction of the blood led to a series of pale colours; the dark red colour was seen at an earlier stage of the infarct. It was in that group that the possibility of pleurisy with subsequent pleural adhesions occurred, and the diagnosis might readily be missed. It had been recorded by Castleman in America that pleural adhesions arising in the scar of an infarct that had become fibrotic had been found at autopsy on patients whose history contained no mention of a diagnosis of pulmonary embolism or infarction. Dr. Rudd thought that the cases described by Castleman fell into the group mentioned by Dr. Morrow—"difficult diagnosis" or "missed diagnosis". Dr. Rudd said that a great debt was owed to Dr. Cummine for the part that he had played in Sydney in stressing the importance and the mode of action of the anticoagulant drugs. Dr. Cummine had followed up their early trials and adoption in America, and had been prominent in Sydney in giving a lead in their therapeutic use. His paper had given a good account of them. Dr. Rudd went on to discuss current views on the process by which the coagulation of blood occurred. Two main theories had been advanced; the first was that of Morawitz and the second was that of Howell. The theories were similar in their fundamentals, but differed in their details; that of Morawitz was more widely accepted. According to that theory, in coagulation of blood a series of changes occurred in which prothrombin, calcium ions, platelets and fibrinogen all played a part. Disintegration of platelets in shed blood led to the formation of thromboplastin. Thromboplastin, in the presence of calcium ions, converted prothrobin to thrombin. Fibrinogen was converted by thrombin to a fibrin clot. According to Howell there occurred a further substance, heparin, which prevented the prothrombin from being changed into thrombin in circulating blood. When thromboplastin was formed in shed blood, it neutralized the heparin, and thrombin was formed from the prothrombin (calcium ions being present). Thrombin thereupon converted fibrinogen to fibrin. Certain objections to Howell's theory stood in the way of its full acceptance, and the theory of Morawitz was the one usually adopted as a working basis. Coagulation, then, was a change brought about by two consecutive enzyme actions, first the conversion of prothrombin to thrombin in the presence of calcium ions and second the conversion of fibrinogen to fibrin by the thrombin so formed. Dr. Rudd went on to say that Lyons, working in the Kanematsu Institute, had some years previously published work in which he stated that a substance termed fibrinogen B occurred in the change of fibrinogen to fibrin. Fibrinogen B was the name Lyons applied to a preparation he made from plasma by a special method. In its ability to clot it differed from fibrinogen prepared by other methods. It was a well-known fact that, in the preparation of pure proteins, the difficulties that one encountered were often due to changes brought about in the proteins by the reagents used in their attempted preparation and purification. That two products differed in their ability to form a clot did not mean that one was more intimately concerned than the other in the coagulation of blood. Dr. Rudd thought that it still remained to be proved that the preparation fibrinogen B was an intermediate substance in the conversion of fibrinogen to fibrin. An attempt had been made to proceed to such proof in the following way. Reagents were prepared which chemically resembled vitamin *K*, and it was shown that the preparation fibrogen B and certain specimens of plasma would both clot with the reagent. The specimens of plasma were obtained from patients in whom thrombosis had occurred or was considered imminent. However, the fact that clotting occurred was not sufficient evidence to establish the identity of two substances as complex as proteins. Further, the preparation of the reagent used in the test was not above reproach from the purely organic chemical point of view. Thus only traces of naphthoquinone could be present in the reagent as finally prepared, but much of the original α or β naphthol. Dr. Rudd thought that until evidence was produced that fibrinogen B was a substance intermediate between fibrinogen and fibrin in the coagulation of blood, and further that the reagent used for its identification was specific, a test for fibrinogen B was unacceptable. It would be better to describe the test as an empirical test for the clotting properties of certain specimens of blood.

DR. F. S. HANSMAN congratulated the speakers on their exposition of the subject. He said that a vote of appreciation was owed to Dr. Cummine for being the apostle of the treatment with anticoagulant therapy of the prethrombotic state. Everybody was in agreement with the importance of the recognition of the prethrombotic state, but some were reluctant to accept the validity of the methods by which the presence of the prethrombotic state was diagnosed. Dr. Hansman wished to confine his remarks to the test for "fibrinogen B" and the value of "positive tests" in relation to susceptibility to intravascular clotting. He regretted that he had to attack the test in the absence of Mr. Lyons, who originated it, but he asked that the subject be debated from the purely scientific point of view because the thing that mattered was the establishment of facts. He said that the work about to be discussed had been carried out at the Women's Hospital, Crown Street, and Dr. J. M. Moyes had played a large part in it. Dr. Hansman then read the following extract from Mr. R. N. Lyons's paper in *The British Journal of Surgery* (Volume XXV, April, 1948, page 362):

> It must be emphasized that the important fact in the interpretation of the test is the presence of fibrinogen B more than the amount, and from the clinical aspect the test is essentially qualitative rather than quantitative.
>
> The reagent used in this test is prepared by dissolving 2 g. β-naphthol in 100 ml. of 50 per cent. alcohol and then exposing this solution to oxygen, when it turns brown. It is not essential to aerate the reagent, but when this is done it appears to increase the rate of gel formation. Since this solution contains 50 per cent. alcohol, both fibrinogen B and profibrin give positive results. However, profibrin only appears when the fibrinogen B concentration has become very high.

Dr. Hansman said that from what he had read out there could be no doubt that Mr. Lyons held that if a gel was obtained "fibrinogen B" was present irrespective of any other consideration; also that profibrin and "fibrinogen B" were two different substances, and that both could be present in the circulating blood in the prethrombotic state. In Dr. Hansman's opinion both of those substances must be considered theoretical' inasmuch as neither had been isolated from fresh blood. He would deal firstly with profibrin. Apitz had done most work on profibrin, and from his published works it would definitely appear that he considered that profibrin was not a substance in its own right but a stage through which fibrinogen passed during its conversion into fibrin. Once fibrinogen was acted upon by thrombin, profibrin was momentarily formed, but its passage into fibrin was inevitable. It could not and did not exist in circulating blood. Mr. Lyons, on the other hand, described profibrin as a substance that existed and circulated in the blood, especially when large amounts of "fibrinogen B" were present; so that Mr. Lyons held a different view from Apitz as to what profibrin was, though Mr. Lyons quoted Apitz as his authority on profibrin. Next with regard to "fibrinogen B": according to Lyons, "fibrinogen B" was converted into fibrin by an oxidative process. In his "test",

that oxidation was brought about by the action of the available oxygen in the naphthoquinone of his reagent. He considered that SH–SH was oxidized to S–S. In 1947 Dr. Moyes introduced "fibrinogen B estimations" as a routine test on out-patients referred to the hospital pathology department for their routine hæmoglobin estimation, Wassermann test *et cetera*. The number of "positive fibrinogen B reactions" varied from day to day. On some days there was a large percentage, on other days a low percentage. As a standard technique was being employed, those results were perplexing; the only variable was the room temperature (Lyons advised that the test should be carried out at "room temperature"). It seemed important therefore to control the temperature, and as it was understood that they were dealing with an oxidative process in relation to blood it seemed logical to use "blood heat temperature", which was 37° C. No positive results were obtained at that temperature. If the test depended on an oxidative phenomenon, the percentage of positive results should have increased over that obtained at room temperatures. The tests were then carried out in the refrigerator at about 8° C., and nearly 100% of positive results was obtained with specimens of plasma taken from apparently normal pregnant women. If they had been dealing with an oxidative process, it would have been expected that the gel formation would be inhibited by a decrease in temperature. In order to test whether oxygen was playing any role whatsoever in the reaction, experiments were carried out with a group of other substances. In each case a specimen of plasma was used that gave a complete gel when tested with Lyons's reagent. A standard technique[1] and a standard room temperature of 15° C. (N.P.L. Standard room temperature) were used. The first substance tried was 1,4 naphthoquinone, a saturated solution of which in 2·5% alcohol contained the same percentage of available oxygen as was present in Lyons's own reagent. No "positive reaction" resulted. By increasing the percentage of alcohol to 25%, a stronger solution of 1,4 naphthoquinone was obtained which contained ten times the amount of available oxygen present in Lyons's reagent. That substance also failed to cause a gel when tested by the standard technique. There was therefore no evidence that they were dealing with an oxidative process. Dr. Hansman and his collaborator then tested whether clotting would take place in the presence of a reducing substance. A freshly prepared 2% solution in 50% alcohol of the well-known reducing substance hydroquinone was used, and that produced gels comparable in every respect to those produced by Lyons's reagent. Similar "positive results" were obtained with other substances, for example, a 2% solution of sodium benzoate in 50% alcohol, and finally when 50% alcohol alone was used as the test substance, solid gels resulted. It was obvious therefore that the gel formation which took place in the plasma of nearly all pregnant women was produced by alcohol acting on some substance present in the plasma. It was more scientific to look for some simple explanation of this phenomenon than to explain it by postulating the presence of two abnormal hypothetical substances. Now fibrinogen itself varied in amount in the plasma from about 0·15 gramme *per centum* to about 0·5 gramme *per centum* in normal people. In pregnancy it had been found in a series of nine cases that the figure could rise to 0·91 gramme *per centum* just before term (Dieckmann and Wegner). If the number of cases examined had been greater, the maximum figure might well have been higher. It was obvious that there was a definite increase of fibrinogen in pregnancy as had long been known and linked causally to the associated increased blood sedimentation rate. However, in spite of this increased fibrinogen and increased sedimentation rate, pregnant women did not show any proneness to intravascular clotting. Fibrinogen was the most labile of all the plasma proteins and was easily converted into fibrin by a number of reagents. Cohn and his school at Boston found that alcohol at low temperatures was a most efficient means of precipitating proteins and they employed it in fractionating the plasma proteins during the war. It seemed logical therefore to conclude that the phenomenon Lyons described as the "fibrinogen B test" was due simply to the precipitation of fibrinogen by alcohol and that a high concentration of fibrinogen and low external temperatures encouraged "positive reactions". In other words it would appear that the test was a measure of the presence of increased amounts of fibrinogen in the plasma. In any condition, such as pregnancy, or in any disease, such as bacterial infections, which was associated with an increased fibrinogen content of the blood Lyons's test could be expected to give "positive results", but that finding *per se* was not an indication of the existence of a prethrombotic state, and in Dr. Hansman's opinion it was unjustifiable to institute anticoagulant therapy on the grounds of a "positive fibrinogen B test". Further research work was indicated, but the test should not be used clinically at the present time.

[1] The standard technique was as follows. Venous blood was oxalated by adding 4·5 millilitres of blood to 0·5 millilitre of 1·1% sodium oxalate solution. Plasma was obtained by centrifuging the oxalated blood. To 0·5 millilitre of fresh plasma in a test tube of one centimetre diameter, 0·05 millilitre of the test substance was added and the tube was shaken and placed in a water bath at 15° C. for five minutes and then examined for gel formation. The tubes were reexamined at the end of thirty minutes.

DR. H. M. RENNIE thanked Dr. Morrow for his masterly survey of the clinical features and differential diagnosis. He also thanked Dr. Cummine for directing attention to the condition. Dr. Cummine had shown that it was necessary to avoid confusion of such vascular lesions with pneumonia and atelectasis. However, it had to be remembered that pneumonia and atelectasis did occur—not all the lesions were vascular lesions. Dr. Rennie asked Dr. Morrow to amplify his statement as to the site of the pulmonary lesions. All lesions were not basal—some might occur in the upper part of the lung. Dr. Morrow had also said that the radiographic appearances might persist for weeks or months or the patient's lifetime. At the present time, when mass radiography was so widely used, that fact should be remembered and the clinical history carefully taken to avoid misinterpretation of unusual shadows.

DR. V. J. KINSELLA, on being called upon by the chairman to contribute to the discussion, said that he wished to ask a few questions. The first was for Dr. Cummine: had he seen deaths follow dicoumarol therapy? Dr. Cummine had mentioned one following heparin therapy. Dr. Kinsella then asked Dr. Rudd to explain that curious macroscopic phenomenon in the early stages of pulmonary infarction, when the infarct was hyperæmic, hæmorrhagic, dripping with blood when cut. Dr. Kinsella said that the same occurred in mesenteric arterial thrombosis, when the lesion would have been expected to be avascular and anæmic. He had never been able to understand it, and was interested that a similar phenomenon occurred in the lungs. Referring to Dr. Hansman's comments, Dr. Kinsella asked, if gel formation following the use of Lyons's reagent was found particularly in a thrombotic state, whether it mattered if the substance involved was called fibrinogen B or just fibrinogen, provided that it gave warning of the thrombotic state in which anticoagulant therapy should be instituted.

DR. B. J. BASIL-JONES said that he was indebted to both speakers for having cleared up a number of difficulties. At the Royal Hospital for Women they used fibrinogen B empirically to detect prethrombotic states. They had found that with different batches of the reagent they obtained very variable results. Some batches gave a gel such as Lyons had described in prethrombotic states, and it seemed that the test would be of considerable assistance. Other batches, however, would not produce a gel, or produced a gel on the least provocation. Dr. Basil-Jones thought that more details about the preparation of the reagent were necessary, as well as more experience in its use. It might give some help after those needs had been met.

DR. E. F. THOMSON said that out of all the discussion about the work that had been done in the past and would be done in the future, one established fact had emerged—that heparin and dicoumarol would control intravascular thrombosis. They had to thank Dr. Cummine for all the work that he had done on that aspect of the subject, and for having given them some definite scheme on which to work. Dr. Thomson did not propose to enter into the discussion of the pros and cons; but to his simple mind there appeared one fact, to which he clung, and Dr. Cummine had consistently tried to point out that he was wrong. Much had been heard at the meeting about the test, and there was no doubt that when Lyons's reagent was added to some plasma a gel was formed, and when it was added to other plasma no gel was formed. It had also been said that the test was a guide, which would help to show whether a patient would suffer from intravascular thrombosis. If those present read Dr. Cummine's articles, the following scheme was laid down—if a patient had a persistent rise in temperature after operation, the blood coagulation time should be estimated twice a day. Dr. Thomson raised the question whether it was possible to determine the coagulation time of the blood. According to Quick it was not possible to do so accurately, and any known test like Lyons's test was purely empirical. But one did estimate the coagulation time of the blood twice a day for patients having a persistently elevated temperature after operation. If the blood coagulation time fell below four minutes, the estimation was repeated at hourly intervals for three consecutive hours, and if the figure remained low heparin was given. But before one gave heparin, one

collected blood on which to perform Lyons's test. Dr. Thomson asked Dr. Cummine whether it was the low coagulation time that was the guide, or whether it was the result of Lyons's test. It seemed that anticoagulant therapy was started before it was known whether the result of Lyons's test was positive or not. Dr. Cummine had said that the platelets disintegrated and formed thromboplastin; Dr. Rudd had said the same. Quick did not believe that that was so. He thought that thromboplastin was in the plasma all the time, and the disintegration of the platelets produced an enzyme which acted on the thromboplastin and turned it into thrombin. Dr. Thomson wondered what was the new clotting factor. Still addressing Dr. Cummine, he then referred to the statement that had been made to the effect that blood became more resistant to the action of heparin when the patient underwent an operation. The test had been worked out on the blood coagulability based on the anticoagulant powers of heparin. Did the mere fact of a person's undergoing an operation make him more resistant to the action of heparin? It seemed that it might be so. The finding might depend a little on the type of anæsthesia and the type of operation. Dr. Thomson then said that frequently in using dicoumarol one noticed that batches of the drug seemed to vary in their action. It was also noticed that the prothrombin index or concentration or time, whichever title was preferred, fell dramatically when only 300 or 400 milligrammes of dicoumarol had been given. He wondered whether that was due to variation of the product, to the patient's being sensitive, or to a combination of both factors. Every now and again one encountered a whole run of people in whom the figure fell suddenly down to 20%; then for a few weeks that would not happen. Dr. Thomson had made inquiries and found that whenever it occurred a new batch of dicoumarol had been used.

Dr. Morrow, in reply, referred first to the site of pulmonary infarcts or emboli. He said that the most common site was the base of the right lung, but the lesions occurred in many other areas. He had an X-ray film of one that had occurred in the apex of the lower lobe of the right lung, but they could be found in various parts of the lung fields. In the case to which he had referred in his paper, in which lobectomy had been performed for suspected carcinoma, the lesion was found in the middle lobe of the right lung.

Dr. Cummine, in reply, said that much criticism had been offered, but in a pleasing way. Those present would remember that, starting from Helen Wright's conception, he had said that in post-operative and post-partum cases there was an increase in the plasma fibrinogen content. Dr. Hansman had brought them back to the original observation—that in the investigation of these people the use of substances of which alcohol was one would demonstrate what appeared to be an increase in the plasma fibrinogen content. An attempt to elucidate this had been made by Lyons, who was a consistent worker in the field of blood fibrinogen, and in his absence one had to accept the supposition that he would be aware of many of the apparent fallacies mentioned. He apparently was not fearful of some of them. When he originally elaborated the test, he used aqueous benzoquinone, not because of its oxidizing properties, but because of its oxidation reduction potential and action upon the sulphydril group. As Dr. Hansman had shown, a variety of substances would produce Lyons's gel formation. It seemed that the essential thing was the use of substances with a certain oxidation reduction potential. Lyons used the quinones because they fell within that range. Those present could consider that, being more or less a specialized worker in the field of fibrinogen, Lyons would know something about the result of heating and thawing plasma and the various reagents. As Dr. Hansman had said earlier, what was said at the meeting would be published, and Lyons would have an opportunity of defending and answering the criticisms. It was best to be convinced neither by him nor by Dr. Hansman, but to remember what might be an analogy. They knew that when Wassermann first described the Wassermann phenomenon, he thought that syphilitic liver had to be used as the antigen. Then some curious investigator used ordinary liver and other substances and still obtained the Wassermann phenomenon. The significance of that reaction was still unknown; but the application of the test to the problems of clinical medicine had gone on like a rolling stone. Lyons's test might have to be modified to meet Dr. Hansman's objections, and those present might have to work out clinically—and they could do that only if all worked at it—whether there was a relationship between the known increase in fibrinogen (whatever it was called) and the occurrence of infarction and embolism. They could only attempt to apply the test and see what its significance was. Dr. Cummine said that he himself had been doing it for almost three years, and being relatively critical of himself and knowing the penalties associated with announcing fatuous and fallacious findings, he felt that there was a close relationship between the phenomenon described by Lyons and intravascular thrombosis. The main thing was to see whether in the teaching hospitals, the centres of most investigation, they could discover some causal relationship between the phenomenon and thrombotic states. In reply to Dr. Thomson, Dr. Cummine said that batches of dicoumarol did vary in their effect; the fact was in no way due to the brand of dicoumarol or to how it was put up. He also had noticed that phenomenon. Much of it could be explained by the individual absorption rate from the alimentary canal. Referring to the prothrombin index, Dr. Cummine said that the question of how it was estimated would require a whole evening's discussion, but it was a very workable test. With reference to Dr. Thomson's remarks about the administration of heparin in relation to blood coagulation time tests, Dr. Cummine said that the reason why the first dose of heparin was given at the same time as the blood sample was taken and before the result of Lyons's test was known was that the test for fibrinogen B had then to be carried out, and if the blood coagulation times were low one could give the patient an injection safely. It was known that hot days did shorten the blood coagulation time; but its estimation was viewed qualitatively and not quantitatively. As Dr. Thomson had said, there was no way of accurately determining the blood coagulation time. But from the estimations that were made one obtained a clinical lead, which was what they as clinicians were seeking. The more complex scientific side of the problem might ultimately be solved. As Dr. Rudd had pointed out, the coagulation of the blood was a series of reactions, not one event. But its clinical estimation could be used in the treatment of a patient. In reply to Dr. Kinsella, who had asked about deaths from dicoumarol therapy, Dr. Cummine said that he had seen three deaths occur. One of the patients had received 1100 milligrammes and had a meningeal hæmorrhage. Another had a retroperitoneal hæmorrhage; in that case the dicoumarol management was incorrect—a wrong prothrombin index had been recorded (the figure was 100% on the fourth day after 600 milligrammes had been given). The third patient had a very prolonged hæmorrhage from the bowel; he died, after nineteen blood transfusions had been given, from what seemed to be metastatic abscesses in the bones and viscera. Dr. Cummine said that he had never had serious trouble when under 600 milligrammes of dicoumarol were given, but he had seen severe hæmorrhages follow 1000 milligrammes.

Dr. Kempson Maddox, from the chair, said that those present had heard a stimulating discussion on a very important problem. Thromboembolic disease caused more deaths than hæmorrhage. Many diseases would be benign were it not that thrombosis or embolism was included in their life history. Dr. Maddox said that he did not agree with all that had been said and regretted that he had not had the opportunity to join in the discussion. A reliable test which would indicate a prethrombotic state would be a great advance. The interest in anticoagulants at the Royal Prince Alfred Hospital stemmed directly from the work of Dr. Cummine and Mr. Lyons. These drugs should only be used in hospitals where adequate laboratory facilities and trained personnel were available. Pulmonary embolism had been robbed of much of its lethal character since the advent of the anticoagulants. They still had much to learn about the mechanism of blood coagulation. Activators and inhibitors operated at each phase of the process. With the great strides in enzyme chemistry currently taking place, they could expect to know much more in the early future. In conclusion, Dr. Maddox called for a vote of thanks to the speakers.

MEDICO-POLITICAL.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on February 2, 1944, Dr. Douglas Thomas, the President, in the chair.

Financial Statements of the Branch.

Dr. J. A. Cahill, the Honorary Treasurer, presented the financial statements of the Branch and of the Medical Society of Victoria for the year ended December 31, 1948. He said that he had very much pleasure in presenting the financial statements for the year ended December 31, 1948. The reasons for this were twofold. Firstly the statements showed that the finances of the Branch were in a healthy condition and that the future could be faced with a degree of confidence that had been lacking in his past presentations.

Secondly, for the first time in the history of the Branch, a requirement of the rules was being met by the submission of a balance sheet. Members would recall that in the previous year Dr. Cahill had promised members that this would be done. This balance sheet set out clearly the assets and liabilities of the Branch, whereas the income and expenditure account of the Medical Society of Victoria and the statement of receipts and payments of the British Medical Association (Victorian Branch) showed the revenue received and the disbursements made in respect of the respective working expense items.

Dealing firstly with the balance sheet of the Medical Society of Victoria, Dr. Cahill said that it would be observed that on the assets side, the Medical Society Hall "Building on Crown Land" had been set down at the arbitrary valuation of £1000. The reason for this was that this building was erected on an inalienable Crown grant revocable by the Crown at will. That being so, the value of the asset as such was not determinable on a realization or other recognized basis. The value of the asset in the building as stated was therefore an arbitrary one. The asset, too, of the books in the library, £2000, had also been arbitrarily fixed for balance sheet purposes, the value of medical works at a given date being difficult, if not impossible, to assess in the present days of rapid advancement in medical knowledge and of slowness and difficulty in getting scientific works printed and made available to readers. The asset, State Savings Bank of Victoria, £2030 7s. 10d., was part of a reserve that had been built up mainly for the purpose of redeeming the debentures on the bulding carrying an interest rate of 5%. Actually this account was in credit £2530 7s. 10d., Commonwealth Bonds to the value of £500 also having been purchased and now being held. The sum of £1500 was credited to the account during 1948. The arbitrary valuation of £1000 for 5000 £1 shares in the British Medical Agency also called for a word or two of explanation. For a long time this Agency had been more or less moribund. Of recent years, however, it had been more active and there was promise in its future if the value intrinsic in private medical practice was not to be disturbed by bureaucratic development. Members would remember that in 1945 the Agency had contributed £1100 towards the purchase of the property at 384-6 Albert Street. Furthermore in the previous year the Agency had presented the Branch with £500 towards the redemption of debentures. The Agency during its existence had on very many occasions contributed to the Branch funds. Dr. Cahill strongly recommended members to avail themselves of the facilities offered by the Agency. By placing their business with the Agency, members might expect good efficient service, and any profits on the service would accrue for professional advantage.

The special funds and accounts as *per contra* shown on the assets side were the totals of the cash balances and value of securities at credit of or held in custody for the funds enumerated on the liability side together with the sum of the imprest cash held at the lease,[3] Spring Street building, the value of the furniture and fittings there at cost, and the value at cost of the freehold property at 384-6 Albert Street. These special funds and accounts assets, it would be seen, added up to £18,741 4s. 10d., while the sum of all the assets amounted to the not unsubstantial total of £28,799 12s. 2d.

As to the items shown on the liabilities side of the balance sheet—the first item was debenture issue. Dr. Cahill would like more than passing notice to be taken of this item. The position was as follows. Twenty-five years previously, a contract to build the hall had been let. To raise funds necessary to meet the cost and subsequent alterations and repairs, 301 debentures had been issued for £7525. Since that issue (twenty-five years) only 89 debentures, totalling £2225, had been redeemed. Of that number, actually 39, totalling £975, had been redeemed during 1948; this had been made possible by a remittance from the British Medical Agency of £500. There were, therefore, still current 212 debentures on which there was a liability of £5300. Of these current debentures, 62 bearing interest at 5% were held by members and 150 bearing interest at 1% by the British Medical Insurance Company. Dr. Cahill proposed, as a business procedure, to apply part of the reserve of £2530 7s. 10d. to redeem the sixty-two 5% debentures and to save an annual interest charge of £77 10s. Dr. Cahill said that members would agree that the nature of the asset, being as he had explained, warranted this common-sense action. Since the original issue of debentures £7321 19s. 4d. had been paid in interest.

The item accumulated account, £4758 7s. 4d., was, of course, the difference between the value of the assets items totalling £10,038 7s. 4d. and the debenture issue item, £5300. They were all enumerated, so it was unnecessary for him to mention them item by item.

Dr. Cahill proposed to deal with the British Medical Association's statement of receipts and expenditure and then with the Medical Society of Victoria's income and expenditure account. Dealing with the British Medical Association's statement first, he said that it would be observed that the income received on the Branch account from subscriptions was £17,020 1s. 3d., and that £1395 8s. 7d. had been received from the Medical Society of Victoria. This latter amount was an adjustment payment to wipe out the bank overdraft on the British Medical Association account at December 31, 1948. On December 31, 1947, there had been an overdraft deficit in this account of £2596 13s. 4d., which was carried forward to 1948.

From this revenue of £18,415 9s. 10d., expenditure to a like amount had been met including the aforementioned bank overdraft of £2596 13s. 4d. The principal items of expenditure were payment to the British Medical Association, London, £2610 4s. 5d., capitation payment to the Federal Council, £1497 15s., subscription allocation to the Medical Society

BRITISH MEDICAL ASSOCIATION—VICTORIAN BRANCH.

Statement of Receipts and Expenditure for Year ended December 31, 1948.

RECEIPTS.

	£	s.	d.
To Balance at December 31, 1947—Cash in Hand	19	0	0
„ Medical Society of Victoria	1,395	8	7
„ Subscriptions	17,020	1	3
	£18,434	9	10

EXPENDITURE.

	£	s.	d.
By Balance at December 31, 1947—National Bank of Australasia, Limited	2,596	13	4
„ Audit Fees	11	11	0
„ Advertising	1	0	0
„ Bank Charges and Exchange	1	0	8
„ British Medical Association—London	2,610	4	5
„ Federal Council of the British Medical Association in Australia—Capitation Fees	1,497	15	0
„ Medical Society of Victoria	5,862	13	6
„ Donation	1	1	0
„ Postages	276	7	5
„ Printing	49	8	1
„ Rebates to Subdivisions	60	13	10
„ Stationery	241	14	10
„ Sundry Expenses	60	4	1
„ Salaries	2,048	0	5
„ Lighting	2	9	11
„ Telephone	16	16	11
„ Travelling Expenses	3	0	6
„ Entertainment	33	3	7
„ Payroll Tax	20	18	10
„ Medical Society of Victoria—Organization Fund	3,020	12	6
„ Balance at December 31, 1948—Cash in Hand	19	0	0
	£18,434	9	10

Compared with the Books and Accounts of the British Medical Association (Victorian Branch) and found to be in accordance therewith.

Melbourne, January 28, 1949.

J. V. M. WOOD & Co.,
Chartered Accountants (Aust.).

MEDICAL SOCIETY OF VICTORIA.

Balance Sheet at December 31, 1948.

	£ s. d.	£ s. d.		£ s. d.	£ s. d.
Debenture Issue—212 Debentures of £25 each carrying interest, some at 5%, some at 1% ..		5,300 0 0	Building on Crown Grant at 426 Albert Street—Arbitrary Valuation		1,000 0 0
Accumulation Account	4,822 1 6		Equipment, Furniture and Fittings at Valuation, December 31, 1948		1,181 10 0
Excess of Expenditure over Income for this Year	63 14 2		Library at Valuation, December 31, 1948		2,000 0 0
		4,758 7 4	National Bank of Australasia, Limited		625 9 0
Special Funds and Accounts, as *per contra*—			State Savings Bank of Victoria ..		2,030 7 10
Debenture Redemption Account	18 10 10		Commonwealth Government Loans at Cost		500 0 0
Property Account 384/6, Albert Street	4,463 19 0		Investments—		
Building Account—Webster House	285 7 6		5,000 Shares of £1 each British Medical Agency Proprietary, Limited, at Arbitrary Valuation		1,000 0 0
Organization Fund	5,217 0 1		16 Debentures 3½% of £100 each Australasian Medical Publishing Company, Limited, at Cost		1,600 0 0
Legacy Fund	1,268 4 2		Sundry Debtors		121 0 6
Food for British Doctors Fund	65 0 0		Special Funds and Accounts, as *per contra*—		
Crawford Mollison Prize Fund	334 8 3		National Bank of Australasia, Limited	1,765 3 11	
Embley Memorial Fund	855 7 2		State Savings Bank of Victoria	7,096 18 10	
Stawell Memorial Fund	891 4 0		Commonwealth Government Loans	5,320 0 0	
Syme Fund	305 15 1		Debentures	505 0 0	
R. H. Fetherston Fund	580 9 7		Imprest Cash	5 0 0	
Income Insurance Fund	2,843 6 2		Furniture and Fittings at Cost	269 2 1	
Annual Meeting (1935) Prize Fund	1,480 9 1		Freehold Property, 384/6 Albert Street, at Cost	3,780 0 0	
Contract Practitioners' Account	132 3 11				18,741 4 10
		18,741 4 10			
	£28,799 12 2				£28,799 12 2

MEDICAL SOCIETY OF VICTORIA.

Income and Expenditure Account for Year ended December 31, 1948.

	£ s. d.		£ s. d.
Subscriptions Allocation	5,862 13 6	THE MEDICAL JOURNAL OF AUSTRALIA	2,020 10 0
British Medical Insurance Company—Grants ..	420 0 0	Library—Books, Journals	603 6 3
Interest	34 19 1	Medical Secretary—Salary	1,528 17 0
Medical Society of Victoria Organization Fund—Grant	200 0 0	Life Assurance Premium	162 17 6
Excess of Expenditure over Income transferred to Accumulation Account	63 14 2	Caretaker Salary	238 6 1
		Audit Fees	12 12 0
		Debenture Interest	165 5 0
		Electricity	128 8 5
		Entertainment	35 15 2
		Legal Expenses	30 9 0
		Office Equipment and Furniture	14 12 0
		Repairs	60 2 0
		Rates and Taxes and Insurance	63 7 8
		Sundry Expenses	47 14 6
		Telephone	73 15 7
		British Medical Association—Victorian Branch	1,395 8 7
	£6,581 6 9		£6,581 6 9

We report that we have audited the Balance Sheet and Accounts of the Medical Society of Victoria, and the Special Funds and Accounts for the year ended December 31, 1948. We have accepted the certificates of the various officers as to the value of certain assets, and in our opinion the above accounts are true and correct according to the books of this Society.

J. A. CAHILL,
Honorary Treasurer.
LEONARD H. BALL,
Honorary Secretary.

J. V. M. WOOD & CO.,
Chartered Accountants (Aust.).

Melbourne, January 28, 1949.

of Victoria, £5862 13s. 6d., salaries, £2048 0s. 5d., and allocation of subscription to the Medical Society of Victorian Organization Fund, £3020 12s. 6d. And for the first time for a number of years there was no overdraft.

From the income and expenditure account of the Medical Society of Victoria it would be seen that the revenue received by the Society during the year was £6517 12s. 7d., including an allocation from members' subscriptions of £5862 13s. 6d., £420 from the British Medical Insurance Company, £34 19s. 1d. interest from certain investments, and £200 from the Medical Society of Victoria Organization Fund. On the other hand expenditure to the amount of £6581 6s. 9d. had been met, the excess of expenditure over income being £63 14s. 2d., which amount was transferred to the accumulation account. The Society's principal expenditure items were *per capita* payments to THE MEDICAL JOURNAL OF AUSTRALIA, £2020 10s.; library for purchase of books and journals, £603 6s. 3d.; salaries, £1767 3s. 1d.; and £1395 8s. 7d. previously mentioned to liquidate the overdraft on the British Medical Association's account. The net result was that the Society's account at the National Bank opened with a credit balance of £625; that during the year £975 of debenture liability had been redeemed, that £1500 had been transferred to the reserve account and that a deficit in the Branch's account was cleared.

That Dr. Cahill thought members would agree was no mean achievement. Nevertheless it was incumbent on him to issue a word of warning. Already they were aware of an increased expenditure in 1949 of £600 for Federal Council capitation (the rate per member had been increased from 15s. to £1 1s.) and of an expense of some £300 for representation at the British Commonwealth Medical Conference to be held in Canada during the year. The spiral of inflation would cause an increase in the cost of services—printing,

stationery *et cetera*—and there were unpredictable commitments ahead following upon the Federal Government's national health enactment. Again Dr. Cahill drew the attention of members to the generous assistance given by the British Medical Insurance Company. The amount received from this source during the year was £420. Members would recollect that with the £2700 the company had given them in 1945 they had been able to purchase the property down at the next corner. Every year they received a substantial subvention from the company. He commended to members the advantages offered by the company when placing their insurances. It was also a pleasure to place on record that in addition to their subscriptions, members during the year contributed £691 to the British Medical Association Legacy Fund, £417 to send food parcels to British doctors, £81 to the Crawford Mollison Fund, £62 to the Federal Medical War Relief Fund and £307 to the Victorian Medical Benevolent Fund.

The report was adopted.

Correspondence.

DRAINAGE TUBE FOR FÆCAL FISTULA.

SIR: Excoriation of the skin of the abdominal wall, following upon a fistula of the small bowel, is a very troublesome complaint. In the treatment of these cases I have for many years been using a special drainage tube to prevent this condition from occurring or to heal it when present. This simple device has invariably proved satisfactory in my hands, and in a number of cases, not only was the patient made more comfortable, but the fistula has healed completely under its use and without any operative treatment. The apparatus consists of a metal tube about three inches long and from one-half to one-quarter of an inch in diameter, attached at an angle of about 30° to a flat or slightly curved metal plate, about three inches square. It is applied in such a manner that, with the patient lying on a slightly inclined plane, or in the Fowler position, the discharge from the fistula will readily pass along the tube and can be caught in a receptacle. A double thickness of lint, with a hole in the centre, and smeared with "Vaseline" on both sides, is placed between the plate and the skin. The apparatus is fixed firmly in position by either a many-tailed or a wide roller bandage. A square of sponge rubber placed between the apparatus and the bandage adds to its efficiency and to the comfort of the patient.

Yours, etc.,

141 Macquarie Street, Sydney, February 16, 1949. P. L. HIPSLEY.

A CASE OF GOLD DERMATITIS SUCCESSFULLY TREATED WITH BAL.

SIR: In an article entitled "A Case of Gold Dermatitis Successfully Treated with BAL" in your journal of February 26, 1949, Dr. Egon Suerth describes a case of rheumatoid arthritis in which gold therapy was employed with some dire results, the one affecting the skin and the other the leucocytes.

After many years of experience in hundreds of cases we have come to the conclusion that the rheumatoid subjects are much more sensitive to gold than the tubercular, and in our rheumatological clinics and private patients have laid down certain rules followed for a number of years.

1. That no case of rheumatoid arthritis will be given any more than 0·05 gramme at a single dose and that the interval between injections should be at least seven days. We noted and published in our first report on gold therapy (1937) that if 0·05 gramme did not produce improvement larger dosage failed to do so, and that toxic manifestations of all varieties, mostly in the skin, were very much more frequent. This dosage is now generally employed in rheumatological practice in England and America in those larger centres where many cases have been studied.

Using maintenance doses of 0·05 gramme "Myocrisin" every six weeks, we have a series of cases which have been maintained on this dosage in very good health, free of all pain and stiffness, without any toxic manifestation, for a period of four years. In the last five years we have seen only one case of severe dermatitis following gold, and that was a very sensitive patient who developed such manifestation after 0·05 gramme "Aurocalcium" (Crookes), necessitating treatment by BAL.

Freyberg's finding may not be out of place here: dosage 100 milligrammes weekly—complications 41%; dosage 50 milligrammes weekly—complications 30%; dosage 25 milligrammes weekly—complications 18%.

2. Sodium ascorbate (1000 units) is mixed in the same syringe with aqueous "Myocrisin" 0·01 to 0·05 gramme and injected at the same time. This may be of help in preventing some serious results, its use, but not this method, being publicized by a continental rheumatologist. Some authorities claim that vitamin C is not protective.

3. In the vasospastic type of cases with pallor and cyanosis, because of the common association of hypoproteinæmia and especially hypoalbuminæmia, methionine two to three grammes daily, calcium gluconate intravenously and liver by mouth are helpful in preventing complications. Since in these cases liver function is disturbed it is essential to keep in mind the toxic effect of gold on hepatic tissues. Methionine as a precursor of cystine and bound up with the mercapturic acids, important in detoxication of metals, is in our opinion valuable both as a preventative agent and in treatment.

4. In the presence of a raised eosinophile count gold therapy is dangerous, and a rising count enforces caution and may necessitate suspension of therapy.

5. If gold produces a leucopenia, pyridoxine 50 milligrammes once or twice weekly is a very valuable remedy, but transfusion must be kept in mind.

We feel that although BAL is a very valuable remedy in diminishing rapidly the toxic effect of gold, strict adherence to low dosage will diminish both the incidence and the severity of deleterious and dangerous reactions.

Yours, etc.,

EVA A. SHIPTON.
L. J. A. PARR.

135 Macquarie Street, Sydney, March 1, 1949.

Royal Australasian College of Surgeons.

PRIMARY EXAMINATION FOR THE F.R.A.C.S.

A PRIMARY EXAMINATION in anatomy (including normal histology) and applied physiology and the principles of pathology will be conducted in Melbourne in November, 1949. The examination will begin with written papers on Monday, November 14, 1949. The examination is reciprocal with the primary examination for Fellowship of the Royal College of Surgeons of England. Each candidate will be notified, by post, of the result at the termination of the examination.

The examination is open to graduates of not less than one year's standing of a medical school approved by the Council for the purpose. Candidates must submit evidence of their qualification, and of the date of acquirement thereof.

Forms of application for admission to the examination may be obtained from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1. The fee for admission, or readmission, to the examination is £15 15s. Australian currency (*plus* exchange on cheques drawn on banks outside Melbourne). The fee must be forwarded with the form of application so as to reach the Secretary at his office not later than October 1, 1949.

COURSE FOR PRIMARY FELLOWSHIP EXAMINATION.

THE Royal Australasian College of Surgeons will conduct a course in anatomy (including normal histology) and applied physiology and the principles of pathology in Melbourne for the primary fellowship examination. The course is open to all medical graduates.

The course will begin in Melbourne on July 25, 1949, and conclude on October 27, 1949. It will consist of six sessions per week. A detailed syllabus is available on application.

Graduates desiring to enrol for this course should register with the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, and at the same time forward the amount of the fee (£31 10s. *plus* exchange on cheques drawn on banks outside Melbourne). Entries for the course close on July 1, 1949. Graduates not resident in Melbourne must notify the Secretary of their Melbourne address immediately after arrival.

NOTICE.

THE following notice was published in the *New South Wales Government Gazette*, Number 27, of February 25, 1949.

CHIEF SECRETARY'S DEPARTMENT, SYDNEY, 25TH FEBRUARY, 1949.

Police Offences (Amendment) Act, 1908, as Amended.

Withdrawal of Authority to be in Possession of Drugs.

It is hereby notified for general information that under the provisions of Regulation No. 25 of the Police Offences (Amendment) Act, 1908, as amended, the authority of Dr. Thomas Patrick Byrne to procure and be in possession of drugs to which the Act applies for the purpose of his profession and to issue prescriptions for such drugs is withdrawn as on and from the 14th March, 1949.

(5990) C. H. MATTHEWS.

WILSON MEMORIAL FUND.

THE following statement is published at the request of Professor A. N. Burkitt.

Some time ago the University of Sydney Medical Society decided to open a fund to create a memorial to the late Professor J. T. ("Jummy") Wilson. W. A. Selle, Esquire, Professor A. N. Burkitt and Mr. I. J. Hunter were appointed trustees. It was suggested that the memorial take the form of a prize in anatomy for medical students. The fund will be closed soon, and any who have not contributed and wish to do so may send their donation to Mr. I. J. Hunter, New Medical School, University of Sydney. Cheques should be made payable to the "Wilson Memorial Fund".

The trustees wish to acknowledge the following:

	£	s.	d.
Previously acknowledged	52	17	0
Dr. F. A. Maguire	15	15	0
Dr. George Bell	10	0	0
Dr. E. W. Fairfax	7	0	0
Professor H. Priestley, Dr. R. E. Nowland, Dr. W. M. Fletcher, each £5 5s.	15	15	0
Dr. D. J. Glissan, Dr. Leslie Allsop, each £3 3s.	6	6	0
Dr. R. W. Richards, Dr. Cotter Harvey, Dr. Garnet Halloran, Dr. Howard Bullock, Dr. R. V. Graham, Sir Charles Blackburn, Dr. R. McD. Bowman, Dr. C. R. Alexander, Dr. E. H. Friedman, Dr. Sydney George, each £2 2s.	21	0	0
Dr. G. Saxby	2	0	0
Dr. W. Moppett, Dr. T. M. Furber, Dr. John Malcolm, Dr. Lindsay Dey, Dr. R. I. Furber, Dr. Archibald J. Collins, Dr. Marjory Little, Dr. H. J. Daly, Dr. H. G. Howell, Dr. H. H. Crowe, Dr. K. Macarthur Brown, Dr. Mary Burfitt-Williams, Dr. Eric Blashki, each £1 1s.	13	13	0
Dr. O. Latham	1	1	0
Total	£145	6	0

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Price, Henry Michael, M.B., B.S., 1948 (Univ. Queensland), Broken Hill and District Hospital, Broken Hill.

Peipman, Eskil Vaino, M.B., B.S., 1942 (Univ. Sydney), 2A Park Street, Clovelly.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Wright, Stanley James, M.B., B.S., 1943 (Univ. Sydney), Queenstown, Tasmania.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Goldberg, Hugo, M.D. (Vienna), registered under the provisions of the *Medical Act*, 1928, of the State of Victoria, 82 Collins Street, Melbourne.

Diary for the Month.

MARCH 14.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
MARCH 15.—New South Wales Branch, B.M.A.: Medical Politics Committee. Ethics Committee.
MARCH 16.—Western Australian Branch, B.M.A.: Annual General Meeting.
MARCH 17.—Victorian Branch, B.M.A.: Executive Meeting.
MARCH 23.—Victorian Branch, B.M.A.: Council Meeting.
MARCH 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
MARCH 25.—Queensland Branch, B.M.A.: Council Meeting.
MARCH 29.—New South Wales Branch, B.M.A.: Council Quarterly.
MARCH 31.—New South Wales Branch, B.M.A.: Annual Meeting.
MARCH 31.—South Australian Branch, B.M.A.: Clinical Meeting.
APRIL 1.—Queensland Branch, B.M.A.: Branch Meeting.
APRIL 5.—New South Wales Branch, B.M.A.: Council Meeting.
APRIL 6.—Victorian Branch, B.M.A.: Branch Meeting.
APRIL 6.—Western Australian Branch, B.M.A.: Council Meeting.
APRIL 7.—South Australian Branch, B.M.A.: Council Meeting.
APRIL 8.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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